Autonomic Prediction of Error-related ERP Components in Perceptual and Conceptual Tasks in Older and Younger Adults

by

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Abstract

In studies of cognitive processing, the allocation of attention has been consistently linked to subtle, phasic adjustments in autonomic control. Both autonomic control of heart rate and control of the allocation of attention are known to decline with age. It is not known, however, whether characteristic individual differences in autonomic control and the ability to control attention are closely linked. To test this, a measure of parasympathetic function, vagal tone (VT) was computed from cardiac recordings from older and younger adults taken before and during performance of two attention-demanding tasks – the Eriksen visual flanker task and the source memory task. Both tasks elicited event-related potentials (ERPs) that accompany errors, i.e., error-related negativities (ERNs) and error positivities (Pe's). The ERN is a negative deflection in the ERP signal, time-locked to responses made on incorrect trials, likely generated in the anterior cingulate. It is followed immediately by the Pe, a broad, positive deflection which may reflect conscious awareness of having committed an error.

Age-attenuation of ERN amplitude has previously been found in paradigms with simple stimulus-response mappings, such as the flanker task, but has rarely been examined in more complex, conceptual tasks. Until now, there have been no reports of its being investigated in a source monitoring task. Age-attenuation of the ERN component was observed in both tasks. Results also indicated that the ERNs generated in these two tasks were generally comparable for young adults. For older adults, however, the ERN from the source monitoring task was not only shallower, but incorporated more frontal processing, apparently reflecting task demands. The error positivities elicited by

the two tasks were not comparable, however, and age-attenuation of the Pe was seen only in the more perceptual flanker task. For younger adults, it was Pe scalp topography that seemed to reflect task demands, being maximal over central parietal areas in the flanker task, but over very frontal areas in the source monitoring task.

With respect to vagal tone, in the flanker task, neither the number of errors nor ERP amplitudes were predicted by baseline or on-task vagal tone measures. However, in the more difficult source memory task, lower VT was marginally associated with greater numbers of source memory errors in the older group. Thus, for older adults, relatively low levels of parasympathetic control over cardiac response coincided with poorer source memory discrimination. In both groups, lower levels of baseline VT were associated with larger amplitude ERNs, and smaller amplitude Pe's. Thus, low VT was associated in a conceptual task with a greater "emergency response" to errors, and at the same time, reduced awareness of having made them. The efficiency of an individual's complex cognitive processing was therefore associated with the flexibility of parasympathetic control of heart rate, in response to a cognitively challenging task.

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Abbreviations

ACC anterior cingulate cortex

BESA brain electrical source analysis

ECG electrocardiogram

EEG electroencephalogram

EPSP excitatory post-synaptic potential

ERN error-related negativity

ERP evoked potential, event-related potential

fMRI functional magnetic resonance imaging

HRV heart rate variability

LP late positivity

OCD obsessive compulsive disorder

RSA respiratory sinus arrhythmia

PD Parkinson's disease

Pe error positivity

PET positron emission tomography

SMA supplementary motor area

SOA stimulus onset asynchrony

VT vagal tone (index)

WCST Wisconsin Card Sort Test

Introduction

One part of the attentional system which is active during the performance of complicated tasks is anterior cingulate cortex (ACC). Located between cortical and subcortical areas, it is uniquely positioned to integrate cognitively and emotionally salient information with motor responses (Banich, 1997). In addition, the ACC has a role in modulating autonomic activity in conjunction with internal emotional states (Devinsky, Morrell & Vogt, 1995). Moreover, it is the most likely generator of the error-related negativity (ERN), a prominent negative deflection in the ERP wave that occurs immediately after an error is made (Dehaene, Posner & Tucker, 1994). The ERN is time-locked to responses made on incorrect trials and thought to reflect neural processes associated with erroneous responding.

In many studies of cognitive processing, the allocation of attention has been associated with subtle, instantaneous adjustments in autonomic regulation (Jennings, Nebes, Yovetich, 1990; Porges, 1995). Such regulation is largely accomplished by the parasympathetic system, which is primarily composed of the vagus nerve and its branches (Hermanowicz & Truong, 1999). Like an automatic car in gear, the heart is designed to "go" unless held back by some kind of brake. Through intermittently slowing the perpetually raring heart, efferent activity of the vagus nerve is the principal means by which the heart responds to changes in the internal or external environment, (Spyer, 1994). Attentional control appears to be more difficult to sustain in later life (e.g. Gaeta, Friedman, Ritter & Chong, 2001). This developmental change might be closely linked to the effects of normal aging or other factors on cardiovascular systems. The focus for this

thesis is to examine cognitive processing related to errors, and its relationship to autonomic responses associated with the heart, in older and younger adults.

Rationale for the Current Study

Numerous researchers have reported relationships between cardiac output and selective attention (Van der Veen, Lange, van der Molen, et al., 2000; Middleton, Sharma, Agouzal et al., 1999; Porges, 1995; Berntson, Cacioppo et al., 1994; Lyytinen, Blomberg & Näätänen, 1992; Jennings, et al., 1990; Mulder, 1985; van der Molen et al., 1985; Lacey & Lacey, 1980; Graham & Clifton, 1966). For example, moment-to moment adjustments in heart rate and the vasculature occur throughout most perceptual and cognitive processes (Jennings et al., 1990) including the maintenance of facts in working memory (van der Molen et al., 1985), regulation of emotion (Porges, 1995) and activation of attention (Richards, 1997; Weber, van der Molen & Molenaar, 1994). Heart rate has been shown to accelerate during the preparation of a response (Mulder & Mulder, 1981) and to decrease in anticipation of the arrival of an expected event (van der Molen et al., 1985). The synchrony of autonomic changes is such that Jennings et al. (1990) concluded that they are induced by the demands of information processing. In addition, there is evidence that individuals with health problems specifically related to cardiovascular function may experience reduced cognitive flexibility. Such individuals have been reported to make significantly more perseverative errors on a common neuropsychological test (Wisconsin Card Sort Test) than those who reported either no health problems or problems unrelated to the cardiovascular system (Dywan, Segalowitz & Unsal, 1992). Thus, cognitive processing in general, and more specifically, the

allocation of attention, appear to be particularly associated with phasic autonomic changes.

Comparisons between attentional allocation and changes in heart rate have been investigated mainly in the context of behavioural outcomes. When it comes to examining heart rate and ERPs, however, relationships have not always been found (e.g., Otten, Gaillard & Wientjes, 1995). This may be because the length of the interbeat interval (the inverse of heart rate) is actually the final outcome of numerous neural inputs to the heart. Relating heart rate, or even heart rate variability (HRV) to another variable amounts to using a gross measure, as it implies making a single quantification of the varying neural inputs that influence cardiac function and relating that unrefined estimate to the other variable. Since this approach has not always yielded relationships between heart rate and psychological correlates, a more specific index of autonomic responsivity was sought for this study.

With recent techniques, it is possible to isolate the various autonomic influences on heart rate that affect temperature, blood pressure and respiration, to some extent. Since control of heart rate on a beat-to-beat basis is mainly parasympathetic (Hermanowicz & Truong, 1999; Spyer, 1994), a measure of parasympathetic control was considered the most useful for the present purpose. Parasympathetic neurons reduce both heart rate and the contractility of myocardial cells through releasing acetylcholine, a neurotransmitter which acts quickly and is quickly deactivated by cholinesterase. Most of the parasympathetic system is made up of the vagus nerve. Research from the past decade indicates that a measure of HRV referred to as vagal tone (VT; see Appendix A)

has shown merit as a physiological index of the parasympathetic system's ability to modulate heart rate to meet environmental demands. The VT index is an estimate of vagal function, calculated over time, based on the specific portion of HRV which is attributable to the small oscillations in heart rate that accompany respiration. This respiratory-linked variation in heart rate is known as respiratory sinus arrhythmia (RSA). It is an estimate of the variance in these periodic oscillations, derived by isolating slower background trends in the cardiac signal and subtracting them from the unfiltered signal (Porges & Bohrer, 1990). A higher VT score indicates more responsive modulation of heart rate. Since the mobilization of cardiac support seems to be intrinsic to the direction of attentional resources, VT was seen as an important and sensitive index of cardiac control.

There is good evidence that some of the factors in attentional control are modulated by the same midbrain and forebrain systems that affect autonomic control of heart rate (Neafsey, 1990; Devinsky et al., 1995; Volkow et al., 2000). In the search for a physiological basis for the problem of declining attentional control, anterior cingulate cortex (ACC) is frequently cited (e.g. Volkow et al., 2000; Luu, Flaissch & Tucker, 2000; Sloane, Hollander, Moss et al., 1999; Turken & Swick, 1999; Mesulam, 1999; Awh & Gehring, 1999; Badgaiyan & Posner, 1998; Vogt, Finch & Olsen, 1992). Among its many functions, the anterior cingulate is associated with attention, motor behaviour, emotional responses and autonomic control. It is subdivided into two divisions that are differentially activated during cognitive and affective processing (Devinsky, et al., 1995; Kiehl, Liddle & Hopfinger, 2000; Bush, Luu & Posner, 2000; Stemmer, Segalowitz,

Witzke, et al. 2001). Its dorsal, cognitive division is involved in the planning, generation and execution of motor commands. Its more rostral, affective division modulates autonomic activity (heart rate, blood pressure, and respiration rate) and endocrine function in conjunction with emotional learning and expressions of internal states. The ACC is engaged during divided attention tasks, when information processing is complicated or difficult and when a response must be selected from among alternatives.

In the past decade, the ACC has been identified as the most likely generator of an important ERP component, the error-related negativity (Falkenstein, Hohnsbein, Hoorman & Blanke, 1990; see also Gehring, Coles, Meyer & Donchin, 1990; Gehring, Goss, Coles, et al., 1993; Dehaene, et al., 1994). This large, negative deflection in the ERP wave occurs in conjunction with the production of incorrect responses. The ERN has been documented in the context of a variety of response time paradigms and across modalities, in conjunction with errors of choice and unintentional action slips. One paradigm that has often been associated with its production is the visual flanker task (Eriksen & Eriksen, 1974). In this paradigm, participants are required to respond to stimuli that are surrounded by either congruent or distracting items, usually by pushing a button or squeezing a dynamometer.

The cognitive operation of interest in this thesis was error-processing. The ERN was selected as a relevant ERP component by which to estimate attentional control. The ERN has been shown to distinguish between groups. For example, elderly adults reliably produce a much smaller ERN component than young adults (Falkenstein, Hoormann, Christ & Hohnsbein, 2000; Band & Kok, 2000; Niewenhuis, Ridderinkhof, Talsma et al.,

2002). Small-amplitude ERNs have also been elicited from individuals with Parkinson's disease (Falkenstein, Hielscher, Dziobek, et al., 2001, but see Holroyd, Praamstra, Plat & Coles, 2002). Patients with obsessive compulsive disorder (OCD) have been shown to produce larger amplitude ERNs, relative to other adults (Gehring, Himle & Nisenson, 2000). Error-related negativities also appear to reflect trait-like differences in individual response style (Pailing, Segalowitz, Dywan & Davies, 2001) such that those whose responses could be characterized as impulsive produce smaller ERNs. Further, the ERN appears to be sensitive to variation in task demands. When instructions stress that accuracy of response is more important than speed, for example, ERN amplitude is larger (Gehring et al., 1993).

Psychologically demanding tasks have been shown to elicit significant changes in cardiac response (Berntson, et al., 1994). Therefore, to explore the cognitive and autonomic changes that occur with age during error-processing, ERPs from error trials in two tasks were examined in the present study–the visual flanker task, and a more complex, conceptual task involving source memory discrimination, which is known to be particularly sensitive to age-related changes in the allocation of attentional control. Eliciting the ERN from the source memory task represents an extension of its use which has not been attempted to date. However, insofar as the ERN reflects an aspect of cognitive processing that is fundamental to error production, one would expect it to be generated by conceptual errors as well as the errors typically elicited by the flanker task. In addition to ERPs from error trials, resting vagal tone was collected before tasks were begun and on-task vagal tone was collected during task performance.

Since autonomic function is modulated to a degree by the anterior cingulate, this raises the possibility that the efficiency of an individual's attentional monitoring will correlate with measures of his or her heart rate variability. If so, it should be possible to find relationships between the amplitude of ERNs in both perceptual and conceptual tasks and measures of vagal tone. If such relations exist, one would expect greater numbers of errors in task performance by those with lower levels of parasympathetic control. To the extent that these systems become less efficient with age, one would also expect reduced ERNs to correlate with poorer parasympathetic control. It was for these reasons that the relationship between autonomic dynamics and attentional control was examined with respect to individual differences and aging. To my knowledge, no study has yet been published which relates age-mediated changes in attentional monitoring, ERN production, and autonomic control in younger and older adults.

Studies of Heart Rate Control and Attention

Parasympathetic control has been a subject of interest in investigations of sustained attention from infancy to adulthood (Richards & Casey, 1991; Richards & Casey, 1992; Richards, 1997; Weber et al., 1994; van der Molen et al., 1996). Among very young infants, Richards and Casey (1991) identified four heart-rate defined phases of information-processing: automatic interrupt (the startle reflex), stimulus orienting, sustained attention and attention termination. In their study, heart rate decelerated for about 5 seconds during orienting to a novel stimulus, was sustained at a low level for between 5 and 20 seconds, and returned to baseline 5-10 seconds after attention termination (i.e. after the stimulus disappeared). These changes are consistent with a

significant increase in, maintenance of and decrease in vagal efferent activity (application and removal of the vagal "brake"; Porges, et al., 1996). Veenhof (1996) has pointed out that the orienting and sustained attention phases are also consistent with William James' (1884) early distinction between two types of attention: involuntary, reactive attention versus attention that can be directed at will. Involuntary orienting must be under sub-cortical control, while voluntary attention must be modifiable by conscious processing.

Infant studies suggest that cognitive developmental outcomes may be predicted by the physiological systems that control heart rate. Fox and Porges (1985) found that mental development within the first year was correlated with a number of physiological measures – including resting vagal tone – taken at 40 weeks gestational age. Infants with average or high vagal tone at term (40 weeks) always had positive developmental outcomes at 8 and 12 months after birth whereas those with low vagal tone had varied outcomes (see also Izard, Porges, Simons, et al., 1991). Huffman and her colleagues (1998) found that individual differences in baseline vagal tone, and the ability to suppress vagal function when necessary, were present 12 weeks after birth. Infants with higher baseline vagal tone have also shown greater dynamic change in vagal activity during a standardized laboratory paradigm. Moreover, those infants who could down-regulate vagal tone most during such a temporary challenge were also those rated by their mothers as being easier to soothe and able to orient for longer periods. The authors concluded that baseline vagal tone subserves the ability to maintain an organized behavioural state and practice with the "vagal brake"seems to promote the development of self-regulatory strategies. (Huffman et al., 1998; Porges et al., 1996). Moreover, delayed maturation of

the vagus nerve in the first 9 months after birth has been implicated as part of the multi-stage process of sudden infant death syndrome (Becker, Zhang, & Pereyra, 1993).

Individual differences in vagal tone tend to stabilize in the second half of the first year of life (Fracasso, et al., 1994), and appear to be firmly established by at least age five (Weber, et al., 1994). By middle childhood, the ability of the heart to selectively respond to attended stimuli has reached adult levels (van der Molen, Somsen & Jennings, 2000). Importantly, the cardiac results of the van der Molen et al. (2000) study have been parallelled by electrophysiological findings. For example, by age eight, unattended visual targets failed to elicit a P300 whereas attended targets routinely prompted P300s (Van der Stelt, Kok, Smulders, et al., 1998). Van der Molen and his colleagues (2000) also found that, even when age effects were removed, more pronounced heart rate deceleration to attended stimuli was associated with less erroneous performance behaviourally (r = -.30).

In 1996, van der Molen et al. investigated the effects of attended and non-attended stimuli on cardiac processes in university students. As in Richards' work with infants, adults' heart rate decelerated while anticipating a rare stimulus, rising again immediately after the stimulus was detected until attention to it terminated. This pattern did not occur in response to equivalent unattended events, which reiterates that higher level processing such as the allocation of attention affects heart function in adults. In a subsequent investigation with infants, Richards (1997) ascertained that heart rate changes actually occur before localization of a stimulus and, moreover, that they index an attentional state which determines whether fixation will shift or not. Thus, cardiac changes appear to be closely linked to the direction of voluntary attention.

Neocortical areas responsible for attention are initially alerted by the reticular activating system, which can simultaneously affect the autonomic system, via the hypothalamus (Brudzhynski, S., personal communication; Kandel, Schwartz & Jessell, 2000). The autonomic system may even be mobilized slightly prior to cortical activation, readying the heart and blood vessels to respond immediately to whatever decision cortical centres make. Cardiac change contributes to the state in which attention can be directed and is therefore a concomitant of attentional processes, ensuing from reciprocal connections among all of these centres.

According to Fallen (2000), the interval between two heart beats is the net outcome of competing bombardment of the heart's pacemaker, the sino-atrial node, by vagal (parasympathetic) and sympathetic neurotransmitters. Berntson et al. (1994) concur, proposing that heart rate is altered via changes in the interplay between parasympathetic and sympathetic firing on the pacemaker. The interplay may be reciprocal, as typically occurs in conditions of physical challenge, such as when the body undergoes orthostatic stress in rising from a sitting to a standing position. However, in situations of psychological challenge, heart rate may change as a result of uncoordinated parasympathetic and sympathetic actions. Furthermore, in other situations, such as during sexual arousal, heart rate may change because these two systems increase or decrease their firing together (see Berntson, Cacioppo & Quigley, 1991, for a review).

In the Berntson et al. (1994) study, young adults were subjected to three mental stress tests: preparing an impromptu speech, performing mental arithmetic, and making a response in time to avoid an unpleasant noise blast in a response time test. Estimates of

both parasympathetic and sympathetic function were taken (respiratory sinus arrhythmia (RSA) and pre-ejection period, respectively; see Appendix A). When cardiac responses for the group were examined, the overall pattern for the group was that parasympathetic activity had decreased as sympathetic firing had increased. However, the correlation between these two systems was negligible, indicating that at the level of the individual, the relative contributions of the autonomic branches to cardiac control varied. Under conditions of mental challenge, the individual differences in cardiac control were large and moreover, remained stable across the three tasks. Thus, it appears to be important to examine relationships between cardiac function and other variables at two levels: at the level of the group and of the individual. Since beat-to-beat variation in heart rate is accomplished mainly by subtle parasympathetic changes (Hermanowicz & Truong, 1999; Spyer, 1994), VT was expected to be a useful measure for ascertaining differences in cardiac function between groups and at the individual level.

The Error-Related Negativity (ERN)

The ERN is a sharp negative deflection that occurs in the ERP wave for trials on which participants have made an error. This component was discovered concurrently in two research laboratories (ERN: Gehring, Coles, Meyer & Donchin, 1990, or, referred to as Ne: Falkenstein, Hoormann, Hohnsbein & Blanke, 1990) by locking averaged ERP responses to a participant's manual response rather than to stimulus onset. It is largest in ERP recordings taken at centrofrontal sites on the scalp (Dehaene, et al., 1994). In brain electrical source analysis (BESA; e.g. Scherg & Berg, 1995), a single dipole in the anterior cingulate was found to account for between 88 and 94% of ERN variance

(Dehaene et al., 1994). Dipole localization must be interpreted cautiously because there is no unique solution to the inverse problem. This means that while the scalp topography of an ERP may be deduced from positive knowledge of a generator's location, the inverse is not true: a given topography may be the result of more than one generator, or a different generator than expected. However, data from event-related fMRI, PET and further ERP studies have provided converging evidence that the anterior cingulate is most likely responsible for generating the ERN (Carter, et al., 1998; Fredrikson, Fumark, Olsson, et al., 1998; Kiehl et al., 2000; Stemmer et al., 2001).

The ERN may be generated in the cingulate sulcus' ventral bank, where the apical dendrites of the pyramidal cells point toward the scalp. Dopamine is thought to have a generally inhibitory effect on neurons in such deep medial prefrontal areas as the anterior cingulate, where it has been shown to reduce the peak amplitude of excitatory post-synaptic potentials (EPSPs; Holroyd & Coles, 2002). When an error has been committed or the results of a voluntary action are worse than expected (i.e., "something is wrong"), dopaminergic activity initiated by the basal ganglia is reduced. Holroyd and Coles (2002) argue that this reduction in dopaminergic activity results in disinhibition of the ventral bank of the cingulate sulcus, producing the ERN. The basal ganglia, and also lateral frontal or prefrontal cortex, have been cited by a number of other researchers as also having roles in the error-monitoring process (Falkenstein, Hielscher, Dziobek et al., 2001; Kiehl et al., 2000; Carter et al., 1998).

Older adults (aged 55-65) have been shown to produce smaller ERNs, despite generating other ERP components such as the N1, P2, and the early P3 of the expected

size (Falkenstein et al., 2000; Band & Kok, 2000). They have also been characterized as having longer response times and less frequent error-recognition as compared to young people. According to Falkenstein et al. (2000), the altered error-processing revealed by the ERN may account for the behavioural differences between older and younger participants, such as the slowed responses and more frequent errors on the part of older adults. (See Appendix B for other conditions affecting ERN amplitude). Such behavioural outcomes have been derived from response time tests and are likely to be of even greater magnitude in any psychologically more challenging test.

Although the ERN was originally thought to indicate error-detection, an ERP component distinct from the ERN – the error positivity (Pe) – has been tentatively identified as a more probable indicator of error awareness (Leuthold & Sommer, 1999; Nieuwenhuis, Ridderinkhof, Blom, et al., 2002; Davies, Segalowitz, Dywan, & Pailing, 2001). Although the function of the Pe is not definitely established (Falkenstein et al., 2000) some researchers maintain that it is a P300 generated to an internal stimulus (namely, the awareness of having made an error), and thus, itself, represents error recognition. In the present study, both the ERN and Pe will serve as indicators of error monitoring in older and younger adults.

Anterior Cingulate Cortex

The ACC has been consistently associated with cognitive processes involving attentional control (Turken & Swick, 1999; Paus, Petrides, Evans & Meyer, 1993; Devinsky et al., 1995; Davis, Hutchison, Lozano, et al., 2000; Bunge, Ochsner Desmond et al., 2001). Blood flow to the ACC is increased when an individual is faced with

performance of a difficult task. Examples of such conditions where greater cognitive control is required include performance of novel tasks, coordination of multiple tasks, selection from between competing responses, and response-monitoring. Conversely, blood flow in the ACC is reduced during less challenging, well-practiced tasks that require little decision-making and simple movements. Interestingly, the ACC seems to be involved in the preparatory decisions which take place before a responsive movement is initiated (Devinsky et al., 1995; Frith, Friston, Liddle & Frackowiak, 1991; Turken & Swick, 1999; Vogt et al.,1992). In addition, PET studies have documented evidence of increased regional cerebral blood flow (rCBF) in the ACC as motor responses are generated (Paus et al., 1993; Frith et al., 1991).

The ACC is well-positioned to affect responses to stimuli because it connects sub-cortical regions that modulate a person's internal emotional state with cortical regions that assist with processing sensory information (Banich, 1997). It is divided cytoarchitechtonically and functionally into areas specific to cognitive and affective processing (Devinsky et al., 1995; Kiehl et al., 2000; Stemmer et al., 2001. See Figure 1). The more caudal, cognitive portion of the ACC has projections that reach the basal ganglia, supplementary motor area (SMA), primary motor area and the spinal cord (Dum & Strick, 1993). Additionally, the ventral bank of the cingulate sulcus is interconnected with dorsolateral prefrontal cortex and tends to be activated concurrently with it, particularly in willed acts (Holroyd & Coles, 2002; Frith et al., 1991). Since the paradigms in the present study require motor responses, activity in the the cingulate's

ventral bank in the more dorsal part (the cognitive division) would be expected during performance of these tasks.

The more rostral affective division has dense reciprocal connections to the amygdala (Devinsky et al., 1995), periaqueductal grey and to autonomic brainstem nuclei (Neafsey, 1990). Activity in all of this circuitry is elicited during the production of affective responses to stressful stimuli (Devinsky et al., 1995; Gemba, Sasaki & Brooks,1986; Schwaber, Kapp & Higgins, 1980). Using fMRI, Kiehl and his colleagues (2000) found that the affective division of ACC was also selectively active during errors of commission, whereas the cognitive division responded on both error trials and correct response trials. Importantly, the affective division (but not the cognitive division) has been implicated in the control of autonomic changes in heart rate, blood pressure, and respiration rate that occur in connection with changing internal emotional states (Devinsky et al., 1995; Neafsey, 1990).

Making an unintended error generally produces an emotional reaction, and thus would be expected to activate both the affective and cognitive divisions of the ACC. Since this area is selectively able to modulate heart rate, it might be expected to alter autonomic function when errors are made. VT should measure how well the parasympathetic branch responds to such modulations. Therefore, since heart rate variation and the generation of the ERN occur concurrently in response to the same event in the same interconnected area of the brain, a relationship between VT and ERN production was hypothesized such that poor parasympathetic control would correlate with the production of small ERNs.

The Effects of Dopamine Loss with Age on the ERN

Relative to healthy, age-matched controls, individuals with mild Parkinson's disease (PD) have been found to generate ERNs with smaller amplitudes in response time tasks (Falkenstein, Hielscher, Dziobek et al., 2001). Moreover, the patients' behavioural data converged with their electrophysiological results: PD patients produced fewer error corrections than their age-matched control counterparts, as well as ERNs with smaller amplitudes.

Falkenstein and his colleagues (Falkenstein, Hielscher, Dziobek et al., 2001) have suggested that, during commission of motor errors, the basal ganglia produce a signal that is conveyed through the limbic system to the ACC, where it produces the error negativity. This resonates with the computational model of Holroyd & Coles (2002) who contend that when the consequences of an action are worse than expected, the mesencephalic dopamine system decreases its firing rate, resulting in a reduction in the amount of dopamine available to the basal ganglia and cortex. This phasic change in firing acts as a negative reinforcement learning signal to the basal ganglia, but is also projected to the ACC. (Conversely, an event-related increase in dopamine acts as a positive reinforcement signal). On the basis of this "error signal", the ACC designates one of a number of neural command centres (e.g. the basal ganglia, orbital frontal cortex, or dorsolateral prefrontal cortex) to take command over the motor system to deal with the situation, and produces the ERN. Furthermore, Nieuwenhuis, Ridderinkhof, Taslma et al. (2002) have used this model to set up computations to mimic both younger and older responses by simulating stronger or weaker phasic alterations of activity in the

mesencephalic dopamine system. The general pattern of their results supported this dopamine hypothesis of altered error-processing in older adults.

There are direct and extensive connections from mesencephalic dopamine cell groups to both prefrontal cortex and the ACC (Bissette, 1996). Output projections of these midbrain dopamine cell groups are densest over the frontal midline, i.e., in ACC and the SMA (Holroyd & Coles, 2002). Whereas D1 receptors are primarily post-synaptic and relate to motor movement, the second major class of dopamine receptors (D2) may be either post-synaptic or serve as autoreceptors (which are located presynaptically), allowing highly integrated control of dopamine synthesis, storage, and release (Bissette, 1996). D2 receptors have also been linked to cognitive measures of mental flexibility and attention (Volkow, Gur, Wang et al., 1998). If the important D2 receptors were lost, control of these operations would be expected to deteriorate, with specific consequences for areas that rely on the availability of dopamine.

Volkow and her colleagues have indeed found reduced dopamine D2 receptor binding in the dorsal striatum (caudate and putamen) and in parts of the posterior cingulate in older participants (2000; see also Bäckman, Ginovart, Dixon et al., 2000; Volkow, Gur, Wang, et al., 1998; Suhara, Fukuda, Inoue, et al., 1991). In the Volkow et al. study (2000), reduced dopamine receptor binding was also associated with lowered glucose metabolism (a marker of brain activity) in both the anterior cingulate and frontal cortex. Moreover, the decline in D2 receptors was accompanied by impaired performance on neurocognitive tasks that involve frontal cortex, suggesting that reduced availability of dopamine had a negative effect on frontal brain function. The correlations

remained significant, even after age had been partialled from the data (see also Bäckman et al., 2000). This suggests that loss of dopamine has an effect on brain function independent of other effects of aging.

If the dopamine hypothesis regarding ERN production is correct, we should not be surprised to see that elderly adults produce ERNs that have smaller amplitudes than those generated by young adults, since dopamine loss is documented to occur with age. Small ERNs are nevertheless remarkable given that they occur in a group that is typically more accuracy-oriented. Ordinarily, such proclivities produce larger ERNs (see Gehring, Himle & Nisenson, 2000). Older adults try to prevent errors and appear to engage in more post-error slowing when erring in cognitive tasks than do younger people (Falkenstein et al., 2000). Despite their care, the behavioural outcomes for older adults in error-processing – more errors, reduced awareness of errors, less frequent error correction, and sometimes complete failure to even determine a correct response (Band & Kok, 2000) – occur. These behavioural outcomes may be partly a function of altered error-processing brought about by the reduction in the amount of dopamine available to frontal cortex and the anterior cingulate (Falkenstein et al., 2000).

Can an ERN be Generated in a Conceptual Task?

An age-attenuation of ERN amplitude has been found in paradigms with simple stimulus-response mappings, such as the Eriksen flanker task. This task typically requires participants to identify the central letter in an array which is flanked on either side by distractors, specifically, by identical letters or different letters. However, the ERN appears to be applicable to a wider range of cognitive activities than simple, speeded response

tasks. To date the ERN has been examined in a few conceptual tasks, including a semantic categorization task (Dehaene et al, 1994), and a colour Stroop task (Gehring et al., 2000). Since it has been more extensively researched using response time tests, however, an ERN generated in the flanker task was thought to serve as a kind of benchmark against which to compare any error negativity from a new task, such as the source memory task. In the flanker task, it was expected that younger adults would generate ERNs with large amplitudes and latencies as is typically reported in the literature, but that those of older adults would be about half as deep (see Falkenstein et al., 2000). ERNs generated in a more conceptual task were expected to be more diffuse. This was because the extra processing required by such a task might involve brain areas whose activity could distort the "electrical signature" of the error-monitoring system (Dehaene et al., 1994). However, one would expect to see the same general pattern with respect to age, i.e., that the ERN of older adults would be smaller relative to those of younger adults. If the ERNs produced by both younger and older adults in a more complex task were less peaked, then the differentiation between their respective ERNs might also be less in the conceptual task than in a standard response time task.

The source memory paradigm is a conceptual task known to be particularly sensitive to age-related deficits in attentional control. In addition to requiring participants to determine whether items are familiar or not, it obliges them to indicate the source of a given memory by distinguishing between two separate sources of familiarity – that attributable to a word's previous occurrence in a study list, and that due merely to a word's repetition within a test list. In previous research (e.g. Dywan, Segalowitz &

Webster, 1998; Dywan, Segalowitz, Webster, Hendry, Harding, 2001; Dywan, Segalowitz & Arsenault, 2002; Veenhof, 1997), it has been found that older adults typically commit more source memory errors than do young adults. Specifically, age differences are found with respect to those non-target words which appear twice in the test list (with six intervening items between repetitions), whereas no age differences are found with respect to the number of correctly identified study words or the number of correctly identified foils. ERPs collected during source memory tasks have also revealed important differences in attentional allocation between younger and older adults. Thus, this task has shown great sensitivity, both behaviourally and electrophysiologically, to age-related changes in attention control. As such, it should reflect age differences in attentional control that are elicited by error-related ERPs. Therefore, the plan of the experiment was to elicit error-related ERPs from younger and older adults in a standard flanker task and a source memory paradigm and compare them.

The Hypothesized Relationship Between Attention and Vagal Function

Since autonomic function is modulated by the ACC, this raises the possibility that the efficiency of an individual's attentional monitoring, as expressed by error-related components, would correlate with measures of his or her heart rate variability. The ability to modulate parasympathetic control in accordance with environmental demands is an important and adaptive feature. As indicated earlier, Richards (1997) and others have proposed that the relationship between heart rate changes and attention is relatively tight.

Therefore, to further delineate the basis of age-related change in error processing, in this study variability in the ERN and Pe would be examined in the context of cardiac function. Resting VT, on-task VT, and a residual measure of VT (VTresid) representing the dynamic change in VT from baseline to task were to be computed from cardiac recordings taken before, during and after task performance from older and younger adults. To the extent that autonomic and attentional systems become less efficient with age, one would expect poorer parasympathetic control to correlate with reduced ERNs and higher vagal tone to relate to larger, that is, more negative, ERN amplitudes. This is based on the assumptions that VT may reflect the general health and responsivity of the cardiovascular system, as well as its close relationship with the attentional system, and that the size of the ERN reflects health of the ACC. Insufficient VT in conditions requiring focussed attention might also be expected to lead to increased numbers of errors. Since both planned tasks demand some level of attentional control, VT was predicted to be associated with the proportion of errors made by participants in both tasks, and between VT and ERP amplitudes, but with the speculation that the relationship would be greater in the source memory task, which would require higher levels of attentional control.

Method

Participants

Thirty-four participants were tested. The data from two have been excluded from all analyses due to technical difficulties in one case and failure to meet inclusion criteria in the other. Sixteen young adults (10 female) and 16 older adults (10 female) participated in this experiment. Younger adults, ranging in age from 19 years to 26 years (M = 20.5, SD =.55), were recruited from an introductory psychology course at Brock University, and received course credit for their participation. Older adults, ranging in age from 61 years to 85 years (M = 72.2, SD = 1.7), were recruited from the local community by newspaper advertisements or word-of-mouth, and received a small honorarium for their participation. All were right-handed.

All older adults reported having achieved some level of post-secondary education, except for two who had not completed high school. One young adult had completed a three-year diploma program and was currently working, but had begun university studies to improve her prospects in her current job. A t-test revealed no difference in education levels between the two groups, t (29) = .04, $p > .9$.

Health and Medical History Questionnaire

On a standard health questionnaire, all participants reported themselves free of psychoactive medications, any serious head injury or concussion and any serious heart ailments. Some of the older participants (n = 5; 32%) reported that they took regular medications to control blood pressure. Those who required blood pressure medications

were included to ensure a more typical older population than is often included in cognition and aging research.

Psychometric measures

Spot-the-Word (Speed and Capacity of Language Processing Test (SCOLP). The SCOLP (Baddeley et al., 1992) is a silent lexical decision task which was administered to all participants to estimate their verbal capacity. The older group had a mean raw score of 52.5 items correct out of 60 (SD = 5.1), which was significantly higher than that of the young adults, (M = 44.19, SD = 4.1), $t(30) = -5.09$, $p < .001$. This difference is typically found when older and younger groups are compared on a vocabulary measure and suggests that the older participants could not be considered to be intellectually disadvantaged relative to the young adults.

Hospital Anxiety and Depression Scale (HADS). The HADS (Zigmond et al., 1983) is a measure of emotional state, and was administered to all participants to assess recent mood. A t-test revealed no differences between groups on either the anxiety scale, $t(30) = .36$, $p > .7$, or the depression scale in this test, $t(30) = -.93$, $p > .3$. Thus, it is unlikely that age differences in mood would account for any differences that might be found in the experimental measures.

Wisconsin Card Sort Test – 64 card version (WCST): The WCST, 64 card version (Grant & Berg, 1948; Kongs, Thompson, Iverson & Heaton, 2000) was administered to all participants as one measure of "executive functions". These would include the abilities to perceive a sorting principle and to shift set (Lezak, 1995). A t-test of the number of successfully completed categories revealed an age effect, $t(29) = 4.6$, $p <$

.001, whereby older adults completed fewer categories ($M = 1.81$, $SD = 1.52$) than young adults ($M = 4.0$, $SD = 1.07$). A t-test of the number of perseverative errors revealed an age effect, $t(27) = -2.7$, $p < .01$, in which older adults committed more perseverative errors ($M = 12.7$, $SD = 5.5$) than young adults ($M = 7.7$, $SD = 4.13$). One outlier was removed from the young adult group before this analysis because he committed more perseverative errors than any other participant, young or old, and because his performance may have been unduly affected by anxiety, indicated by a clinically high anxiety score on the HADS. Thus, although there was no difference in education between young and old, and a difference in vocabulary level in favour of older adults, the differences in WCST performance in favour of younger adults suggested that the older adults in this sample had the attentional control problems usually associated with aging. Such difficulties are usually associated with a decline in frontal lobe function. It was important to establish the group difference in attentional control in order to interpret any group differences in ERP and cardiac variables.

Procedures

Participants were welcomed at the lab, given a brief tour and seated in a testing room where the procedures were explained to them. Then they were asked to complete the health questionnaire, mood index, vocabulary test and WCST. Participants were then taken to a dimly lit, electrically shielded recording room where an elastic cap (Electro-cap), heart electrodes and respiration strap were applied while the participant was seated comfortably. A baseline recording of heart rate and respiration was taken before

beginning the first task, after the first task, and at the end of the session when all tasks were completed.

The Flanker Task

This visual discrimination task is a classic paradigm for investigating the effects of response competition and interference (Eriksen & Eriksen, 1974), and has been used extensively in the past decade in the investigation of the ERN. To do the flanker task in this experiment, participants had to decide whether the central letter in a 5-letter array was an H or an S, and press a key corresponding to the appropriate letter. The surrounding letters could be identical to the central letter (HHHHH or SSSSS, congruent condition, 80 trials each) or different from it (HHSHH or SSHSS, incongruent condition, 160 trials each). Congruent and incongruent trials were randomized.

Flanker Procedure. Participants were seated comfortably about 45 cm from the computer screen. EEG was recorded continuously during the task. Stimulus duration was 250 ms with stimulus onset asynchrony (SOA) being 1250 ms. The assignment of the letters to each hand was consistent for each participant and counter-balanced across participants. Participants were given two short breaks, after 160 trials and after 320 trials. During these few seconds no stimuli appeared on the computer screen. There were no practice trials for this task.

The Source Memory Task

The source memory task was presented as two blocks of trials in order to ensure sufficient responses for averaging ERPs. The flanker task was presented during the break between the two blocks of the source memory task. The stimulus set consisted of 200

5-letter words, presented in white lower case letters about 1 to 1.5 cm in height, on a black screen. Mean frequency of the words was 126 per million, according to the Kuçera and Francis (1967) norms. They were divided into 8 lists of 25 words, balanced for frequency of occurrence. At test, four separate lists were presented in each block: one list served as study items, one other as lag items (presented twice, first occurrence and second occurrence) and two others as foil items. There were 6 practice trials at the start of the test phase. Thus, each block consisted of 25 study items and 131 (6 for practice) test items.

The lists were rotated sequentially as participants were tested so that differences in performance could not be due to the characteristics of a particular word list or order of presentation. Word type (study, lag foil) was pseudo-randomized to prevent selective fatigue effects.

Source Memory Procedure. Each word from a study list of 25 items was presented for 5 seconds. Participants read each word aloud and were asked to attend to it. They were warned that they would be asked to recognize it later. In the test phase, participants were asked to identify each study word by pressing a key representing "Yes" if the word presented was from the study list or "No" if it was not.

Participants were informed before the test phase began that some of the new words would be repeated, but that their primary task was to classify each word in the test list as to whether it was a study word or not. On completion of the test phase, they were given a simple recognition test, i.e., a list of printed words which includes all words from the study list interspersed with frequency-matched new words not yet seen. They were

asked to circle those they remembered from the original study list. The number of correct hits and false positives in the paper-and-pencil recognition test provided an index of general recognition ability when the source memory lures were not present.

To ensure enough trials from which to form ERP averages, the entire study-test procedure was repeated using new word list, the two sessions, as was indicated above, being separated by a rest period and the flanker task. Scheduling the tasks in this manner was done to reduce the likelihood of words from the first session interfering with the second session.

Physiological measures

To assess general physiological reactivity, the electrocardiogram data (ECG) were continuously collected throughout the tests of cognitive function and for three minute periods at three baseline points, before the first lag task (Baseline 1), immediately after the first lag task but before the flanker task (Baseline 2), and at the end of the session, when all three tasks had been completed (Baseline 3).

Electrocardiogram (ECG). The ECG entails placing two electrodes on the participants' chest, about 2.5 cm below the left clavicle and about 5 cm below the top of the sternum, at the midline. Since the cardiac signal is much larger than EEG signals, the usual hardware gain of 10,000 was reduced to 1000 by an attenuator. Calculation of individual resting vagal tone was made offline from the ECG data according to Porges' (1985) technique for detrending and filtering the heart rate signal.

Residual measures of change in vagal tone, derived from regressing baseline vagal tone from the vagal activity calculated from cardiac data collected during

performance of three tasks were analysed as estimates of dynamic vagal change. ECG data were not analysed during the study phase of the lag task because speaking the words aloud during this phase would have disrupted breathing patterns. This is known to produce confounds in vagal tone measurement because part of the vagus nerve regulates vocal intonation (Porges 1995).

Electrophysiological Recording (EEG).

The acquisition and stimulus presentation program was InstEP (InstEP System, Inc., Ottawa, Ontario). During performance in both the lag and the flanker paradigms, EEG was recorded from 36 scalp electrode sites (Fpz, Fz, FCz, Cz, Pz, Oz, AF3, AF7, Fp1, FC1, F3, FC5, F7, CP1, C3, CP5, P3, PO3, P7, T7, O1, AF4, AF8, FP2, FC2, F4, FC6, F8, CP2, C4, CP6, P4, PO4, P8, T8, O2) according to the extended international 10/20 system. (These sites are illustrated in Figure 2). The electrodes are embedded in an elastic cap (Electro-cap International, Inc., Eaton, Ohio). All electrodes were re-referenced offline to an average of the signals at the two ears with a right mastoid ground. Impedances were maintained below 5 kOhms.

Eye movements were recorded from two electrodes place above and lateral to the right eye and regressed out of the recorded signal offline. For the numerous correct responses to flanker trials, rejections for movement artifact were set at $\pm$ 75 μV for all channels and any such trials deleted by an automated procedure. For all error responses and for the correct responses in the source memory paradigm, each single-trial ERP waveform was visually inspected before averaging and trials with excessive movement artifact were rejected manually. The amplifier gain was 10,000 (Sensorium, Inc.), and

the EEG was digitized with a 12-bit ADC processor. Signals were sampled at a rate of 256 points per second. For error trials in the flanker task and source memory task, 1000 ms epochs were extracted, from 600 ms before the response until 400 ms after it. For correct source memory trials, ERP epochs were 2000 ms, including a 200 ms baseline. For both tasks, the data from each epoch were filtered at .16-30 Hz. To quantify peak amplitude and latency of ERP waveforms such as the ERN, a computer-assisted hand-scored peak analysis program (Segalowitz, 1999) was used.

EEG data from the flanker task were averaged with respect to response onset. In this task, ERN amplitude was defined as the difference between the most negative value in the 200 ms following the response and a baseline defined as the signal in the period -600 to -400 ms preceding the response. Pe amplitude was defined as the difference between the most positive value in the period 140-350 ms post-response and the baseline period -600 to -400 ms preceding the response. The EEG data from the source memory task were averaged for each participant both with respect to stimulus onset (for Late Positivity (LP) responses to the stimulus) and response onset (for the error-related components). In this task, ERN and Pe amplitudes were defined as they were in the flanker task, but with reference to the signal in the 200 ms preceding the response. The reasons for the different baselines are explained in the next section.

Results

General Analysis Procedures

The analyses presented here have been corrected for violations of Mauchly's Test of Sphericity, where necessary, using the Huynh-Feldt correction for degrees of freedom and F-statistics. Refer to Appendix D for analysis summary tables. Interpretation of any ERP interactions with site should be made with caution as the electrophysiological data have not yet been scaled according to the method of McCarthy & Wood (1985) to confirm that there were no specious interactions between condition and site.

The Flanker Paradigm

Behavioural Responses. The mean number of errors to the congruent and incongruent flanker stimuli are presented in Table 1. Since the focus of this study concerned the commission of errors and its relationship to error-related processing, the total per cent of error responses from the flanker task were submitted to a 2 x 2 mixed model analysis of variance (ANOVA) with condition (congruent vs. incongruent trials) as the within-group factor and age (older vs. younger adults) as the between-group factor. Older adults ($M = 11\%$) made more errors than younger adults ($M = 7\%$), $F(1, 27) = 5.11$, $p = .03$, $\eta^2 = .16$. As may be seen in Figure 3, participants (from both groups) were more likely to make errors in response to incongruent trials ($M = 10\%$, overall), where the flanking letters differed from the central target letter, than to congruent trials ($M = 6\%$, overall), where the flanking letters were identical to the target letter, $F(1, 27) = 37.0$, $p < .001$, $\eta^2 = .58$. While not central to the present study, these findings are consistent with other reports in the literature indicating the increased level of task difficulty that attends

incongruent trials (e.g., Falkenstein et al., 1991; Pailing et al., 2002; Niewenhuis, Ridderinkhof, Talsma, et al., 2002). Older adults made significantly greater percentages of errors on congruent trials than younger adults $t(27) = -2.47$, $p = .02$, whereas the numbers of incongruent trials between groups failed to reach significance, $t(27) = -1.71$, $p = .10$. Behavioural data from two older adults were omitted from these analyses due to erratic responding.

Response Times. Response times for correct trials were analysed in the same way as above and are presented in Table 2. As expected, older adults responded more slowly ($M = 547$ ms) than did younger adults ($M = 490$ ms), $F(1, 27) = 10.92$, $p < .003$, $\eta^2 = .29$. Participants responded more quickly on congruent trials ($M = 504$ ms) than incongruent trials ($M = 531$ ms), $F(1, 27) = 104.9$, $p < .001$, $\eta^2 = .80$. This is also consistent with earlier findings (Falkenstein et al., 1991; Pailing et al., 2002). Eriksen and Eriksen (1974) originally reported that with respect to congruent flanking letters, incongruent flanking letters were more distracting and exacted a higher cost to response times. There was also an interaction between congruency condition and group, $F(1, 27) = 6.45$, $p = .02$, $\eta^2 = .19$, indicating that young adults were proportionally faster than older adults on correct congruent trials (see Figure 4).

Electrophysiological Responses. The ERP responses of younger adults to correct and error trials are presented in Figure 5. The P300, error-related negativity (ERN) and error positivity (Pe), were identified in response-locked ERP averages of incorrect trials for each group and are presented in Table 3. As illustrated in the figure, the P300 is a positive deflection that occurs around the time of the response (0 ms). The ERN is a sharp negative deflection that occurs within 100 ms of the response, followed by the Pe, a

broad positive component that spans a period from about 140-350 ms. Figure 6 depicts the same ERP components for the older adults, where it would appear that the waveforms associated with correct and error trials are less differentiated, especially from 100 to 400 ms post response. As may be seen in Figures 5 and 6, the P300 component is response-locked and present in both correct and incorrect trials, whereas the ERN and Pe appear only in the error trial waveforms.

The amplitude and latencies for all components in this task were determined in relation to a baseline ranging from -600 ms to -400 ms prior to the response. This baseline was selected because it avoids including the positive contribution of the P300 which occurs around the time of the response. Since, in the congruent condition, there were few error trials (younger $M = 6$; older $M = 12$), it was necessary to average the error waveforms from congruent and incongruent trials in order to ensure maximal numbers of trials for each average waveform.[1]

The Flanker P300. The P300 component is common to both errors and correct responses and is thought to reflect registration of the stimulus. While apparently slightly

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[1] Waveforms to incongruent and congruent error trials have been found to be very similar (see Pailing et al., 2002). To determine whether this were true for the present data set, the amplitude data for the ERN and Pe were submitted to separate 2 x 4 x 2 mixed model ANOVAs with condition (congruent vs. incongruent) and site (Fz, FCz, Cz or Pz) as within-group factors and age (older vs. younger) as the between group factor. For the ERN, there was no effect of condition, no interaction between condition and group and no three-way interaction with site. For the Pe, a significant main effect of congruency condition was revealed, $F(1, 23) = 9.25$, $p = .006$, $\eta^2 = .29$, such that the Pe to congruent errors was larger than to incongruent errors, but it did not interact with group. However, because the waveforms cannot be conveniently divided between ERN and Pe components, both incongruent and congruent trials were included in the averaged waveforms for each group in order to produce waveforms that were as reliable as possible. Reported analyses concerning each component are of mean ERN or Pe amplitudes collapsed across congruency conditions.

smaller for the older group (older: $M = 3.56$ μV vs. younger $M = 4.89$ μV), the P300 on errors trials as measured in the flanker data was not statistically different across groups, $F(1, 30) = .58$, $p = .45$, $\eta^2 = .02$. This negative finding is important, because it implies that any age differences in the error-related components (ERN and Pe) in this sample would not be attributable to a general decline in overall EEG power due to aging. The P300 showed an effect of site, $F(3, 90) = 5.57$, $p = .01$, $\eta^2 = .16$, which was superceded by a site by group interaction, $F(3, 90) = 7.34$, $p = .004$, $\eta^2 = .20$. Post-hoc analyses indicated that for the younger adults, the P300 had a central-posterior distribution, being largest at Cz and Pz. For the older group, the P300 appeared to be largest at FCz but analyses indicated that its amplitude did not differ across sites. These data form a pattern similar to one found by Niewenhuis, Ridderinkhof, Talsma, et al. (2002), who also reported a highly significant interaction between age and site that is typical of the hyperfrontality usually associated with age (e.g., Dywan et al., 2002; Friedman, 2000. Figure 7 illustrates the response-locked P300 to correct trials. There was no statistical difference between the P300 to correct and incorrect trials).

The Flanker ERN. An analysis of ERN amplitudes from congruent and incongruent trials indicated no effect of congruency condition, $F(1, 25) = .71$, n.s. and no interaction between condition and group, $F(1, 25) = 2.06$, n.s. Thus, the ERN amplitude data could be collapsed across congruency conditions. Figure 8 illustrates the collapsed and averaged waveforms to error trials for older and younger adults. The ERN, shown most clearly here by the younger adults, is a prominent negative deflection, beginning around the time of the response and peaking about 70 ms later. These ERN amplitudes were submitted to a 4 x 2 mixed model ANOVA with site (Fz, FCz, Cz or Pz) as the

within-group factor and age (older vs. younger adults) as the between-group factor. The ERN of older adults (M = -2.1 μV; maximum, Cz = -3.09 μV) was shallower (i.e., less negative) than that produced by the younger adults (M = - 8.06 μV; maximum, FCz = -11.57 μV), $F(1, 23) = 12.05$, $p = .002$, $\eta^2 = .34$. The ERN amplitudes are presented in Table 3. There was a significant effect of site, $F(3, 69) = 23.09$, $p < .001$, $\eta^2 = .50$, indicating that the ERN was most negative at fronto-central sites FCz and Cz. Post hoc comparisons indicated no difference between FCz and Cz but at Fz and Pz the ERN waveform was significantly shallower. The site by group interaction was significant, $F(3, 69) = 11.08$, $p < .001$, $\eta^2 = .34$. Post-hoc tests confirmed that for younger adults, the ERN activation was most negative at FCz and dissipated at Fz and Pz. For the older adults the ERN was more diffuse, with a slightly more central distribution (maximal at Cz). The results reported here are from an analysis that used only 9 young subjects. (These nine were selected for the purposes of cross-task analyses, as they made sufficient errors in both tasks to form reliable ERN waveforms). This analysis of ERN amplitudes was repeated using all 16 young subjects, with no differences in the outcomes.

Another standard approach for calculating ERN amplitude is to create a difference wave (see Figure 9). The difference wave is calculated by subtracting the waveform to correct trials from the waveform to incorrect trials. ERN data from all 32 participants were analysed. Submitting these peaks to an analysis of variance revealed a significant effect of age group, $F(1, 30) = 9.99$, $p = .004$, $\eta^2 = .25$, and of site, $F(3, 90) = 23.25$, $p < .001$, $\eta^2 = .44$. The interaction term was also significant, $F(3, 90) = 4.05$, $p = .03$, $\eta^2 = .12$. Post hoc analyses indicated that the difference wave ERN was maximal at FCz and Cz for both groups but became comparatively less negative at Pz for the younger group.

Since the difference wave contained no ERP effects related to processing of correct trials, and analyses of its components produced the same pattern of results as the averaged wave, it was concluded that the ERNs derived from direct measurements were the results of error processing (see Table 4). Any further analyses were conducted on the standard ERN.

Finally, whenever one is faced with amplitude differences in ERP components between conditions or groups, one has to determine whether these differences can be accounted for by small latency differences between trials known as jitter. If the ERN peak in each trial for a given participant occurred with slightly different latencies, when the peaks were averaged together to obtain ERPs for scoring, the resulting waveform would be comparatively shallow. This was a concern because in the present data set the variability in error response times (i.e., the standard deviations) appeared to be greater for the older group (M = 126 ms) than for the younger group (M = 71 ms). A t-test of the standard deviation for incongruent trials, which represented the bulk of the errors (1004 out of 1286 error trials), confirmed that the age difference was significant, $t(27) = -3.33$, $p = .003$. Since the ERPs are time-locked to the responses, this variability could have had implications for the average.

To see whether such differential variability could be a source of waveform attenuation for the older adults, the standard deviations of response times were correlated with the amplitude of the ERN for each group. The results ($p > .2$ for every site) indicated that no correlations between the standard deviation for response times and ERN amplitudes were significant for either group. Thus, one could conclude that the relative

shallowness of averaged ERN components for the older group was not attributable to jitter.

The Flanker Pe. The ERN was followed by a lengthy positive deflection, the Pe. The Pe for the younger group (M = 10.55 μV) was larger than for the older group (M = 5.3 μV), $F(1, 30) = 8.52$, $p = .008$, $\eta^2 = .27$. There was also an effect of site, $F(3, 69) = 15.27$, $p < .001$, $\eta^2 = .40$. Post-hoc comparisons indicated that the Pe was most positive at fronto-central sites, FCz and Cz. A site by group interaction was also significant, $F(3, 69) = 17.78$, $p < .001$, $\eta^2 = .44$, such that for the younger group the Pe was central-posterior, being maximal at Cz, but for the older group it was more diffuse and slightly more frontal. The results reported here are from an analysis that used only 9 young subjects. When repeated using all 16 young subjects, the outcomes were no different.

Pe amplitudes from the difference wave using data from all 32 participants were submitted to a mixed model analysis of variance. For the difference wave Pe, there was an age effect, $F(1, 30) = 7.77$, $p = .009$, $\eta^2 = .21$ and an effect of site, $F(3, 90) = 31.83$, $p < .001$, $\eta^2 = .52$. The interaction between age and site was also significant, $F(3, 90) = 17.37$, $p < .001$, $\eta^2 = .37$. Post hoc analyses indicated that the Pe for the older group was of similar amplitude across all four midline sites, whereas the Pe deflection for the young was large at Cz and Pz relative to the more frontal FCz and Fz. Thus, the results of the analysis of the difference wave Pe were similar to those from the averaged wave, confirming that the Pe in the averaged wave was associated with error-processing.

Since there was a significant difference in the response time standard deviations for older and younger adults, the possibility that latency jitter was responsible for the flatness of the Pe component in the ERP wave of older adults also needed to be assessed.

As was the case for the ERN, a correlational analysis of Pe amplitudes with the response time standard deviations was carried out. None of the correlations was significant ($p > .2$ at every site). It was concluded that the shallowness of the elder Pe component was not attributable to jitter.

In summary, the behavioural and ERP results outlined here suggest that behavioural performance and electrophysiological responses in this study were typical for flanker paradigms using letter stimuli. They indicate that responses to incongruent trials are more challenging in that they are more likely to elicit errors and slower responses than congruent trials. Also, commission of errors elicits two electrophysiological responses which are not present for correct trials. These are the ERN (which occurs within 100 ms of the error response) closely followed by the Pe. Older adults make more errors and are slower in responding than younger adults. Furthermore, when they make errors, they generate smaller ERN and Pe components (see Figure 10), but not because of an overall decline in EEG power or increased variability in their response times.

The Source Memory Paradigm

Behavioural Responses. There have been numerous studies confirming that production of the ERN and Pe occurs in association with error processing in the flanker task (e.g., Falkenstein et al.,1990; Gehring et al., 1993; Stemmer et al., 2000; Luu, Flaisch & Tucker, 2000) as well as other work that has demonstrated the existence of age differences in the ERN and Pe similar to those reported here (e.g. Gehring & Knight, 2000; Falkenstein et al., 2000, Falkenstein, Hoorman & Hohnsbein, 2001; Niewenhuis, Ridderinkhof, Talsma, et al., 2002). However, there was still a question as to whether these ERP error-related components could be elicited in a more complex task requiring a

different kind of attentional control, and if they were elicited, whether these effects would also be susceptible to the effects of age. To answer this question, we selected the source memory paradigm, which has been shown to be particularly sensitive to age-related change in attentional control. This task requires participants to distinguish between the familiarity associated with words previously seen in a study list and that associated with non-target words which are merely repeated in the test list. Age differences are typically found with respect to the repeated words, but not to correctly identified study words or foils.

In order to determine whether performance on the source memory task in the present study was typical, a measure of performance was calculated for each type of stimulus (study, lag or foil) based on the proportion of each to which participants responded "yes" (see Table 5). The mean proportions of "yes" responses for study, lag and foil words were submitted to a 3 x 2 mixed model ANOVA with word type (study, lag or foil) as the within-subjects variable and age (older vs. younger adults) as the between-subjects variable. A main effect of age group was significant, $F(1, 30) = 7.1$, $p = .01$, $\eta^2 = .19$, such that the older group ($M = .40$) was more likely to say "yes" overall than the younger group ($M = .29$). As expected, there was a significant effect of word type, $F(2, 60) = 165.8$, $p < .001$, $\eta^2 = .85$, such that study items had the highest proportion of "yes" responses ($M = .59$), followed by lag items ($M = .33$) and foils ($M = .12$). Additionally, there was an interaction between word type and group, $F(2, 60) = 30.66$, $p < .001$, $\eta^2 = .51$. Post-hoc comparisons indicated that while both groups selected similar proportions of study words correctly (younger $M = .63$; older $M = .56$), and made similar proportions of errors on foil items (younger $M = .08$; older $M = .15$), they differed

in the proportions of errors they made on lag items (younger $M = .16$; older $M = .49$). Thus, with respect to the younger group, the older group made more than three times the number of errors on the non-target, repeated, lag items, but identified study words and foils about as well as younger adults. These data are presented graphically in Figure 11. Correlational analyses revealed a relationship approaching significance between lag errors and the number of categories achieved in the WCST ($r = -.43$, $p = .053$), such that greater numbers of errors were marginally associated with completion of fewer WCST categories, primarily for the older adults (see Figure 12). Thus, reduced attentional control as seen in the WCST was also reflected in greater numbers of source errors on the part of older adults.

Response Times. Response time data are presented in Table 6. ANOVA results indicated that, as expected, the responses of older adults were slower ($M = 1238$ ms) than younger adults ($M = 1033$ ms), $F(1, 30) = 4.8$, $p = .04$, $\eta^2 = .14$, but no effect of word type nor interaction between word type and group was found.

ERP Responses (Stimulus-locked). The averaged stimulus-locked ERP amplitudes for older and younger correct and error trials are presented in Table 7. They include stimulus-locked ERP data from those participants with enough errors to provide sufficient trials to form a stable ERN (minimum 7 trials).

The Source Memory LP. The component that is believed to reflect evaluation of the stimulus in the source memory task is the Late Positivity (LP). This is a long, positive-going deflection lasting from about 400 ms to 800 ms post stimulus onset (refer to Figure 13). The LP is thought to reflect the amount of attention allocated to the correct

detection of target or non-target events and has traditionally been identified in correct trials than have been stimulus-locked.

To compare ERP responses across the different experimental conditions, stimulus-locked averages of EEG were prepared for each word-type (study words, lag words, and foils). These are illustrated in Figure 13. To ensure sufficient trials to form a reliable LP, correct trials from the two blocks of the source memory task were combined. (Sixteen are usually considered minimal for adequate signal to noise ratio). Based on visual inspection, an estimation of the LP for each site was made by computing the average area between 450 and 850 ms post-stimulus onset for each word-type. Even under these conditions, three participants had what was considered insufficient correct trials in the averages to score a reliable LP (< 14 trials). When the main analysis was carried out with and without these participants, however, there were no differences in the outcomes. Therefore, LP amplitude data from all 32 participants were included in the waveforms and analyses reported.

The average LP areas from correct lag-word trials from Block 1 and Block 2 combined were submitted to a 3 x 4 x 2 mixed model ANOVA with word-type (study, lag or foil) and site (Fz, FCz, Cz or Pz) as within-group factors and age (older vs. younger) as a between-group factor. There was no main effect of age, indicating that there was no overall difference in EEG power between groups. The analysis revealed, however, an effect of word-type, $F(2, 60) = 8.74$, $p < .001$, $\eta^2 = .23$, which was superceded by a word-type by age group interaction, $F(2, 60) = 4.13$, $p = .02$, $\eta^2 = .12$. Pairwise comparisons revealed that the younger group produced the greatest LP to target study words ($M = 1.85$ μV), relative to lag words ($M = .78$ μV), and foils ($M = -.88$ μV). In contrast, the older

group produced the largest LPs to lag words (M = 1.80 μV), relative to study words (M = 1.37 μV), and foils (M = .91 μV). The pattern of LP size in the older adults replicates Dywan et al. (1998, 2001, 2002) such that for younger adults, responses to study words were clearly distinguishable from responses to lag words and foils but that for older adults the LP amplitudes were less differentiated on the basis of a targeted relative to a non-targeted response.

The analysis also revealed a main effect of site, F (3, 90) = 7.21, p = .003, η^2 = .19, which was superceded by a site by group interaction, F (3, 90) = 16.43, p <.001, η^2 = .35, such that the younger group's largest LP amplitude was at Pz (M = 2.19 μV), followed distantly by the other three midline sites, Cz (M = .68 μV), FCz (M = -.27 μV) and Fz (M = -.27 μV). In contrast, the amplitudes for the older group were similar at all four midline sites, Fz (M = 2.18 μV), FCz (M = 1.32), Cz (M = .76 μV) and Pz (M = 1.19 μV). These site differences across group did not interact with word type.

To summarize, the pattern of these results replicate those of previous studies (e.g., Dywan et al, 1998, 2001, 2002). To the extent that the LP reflects attentional allocation, it would appear that younger adults are able to allocate attention to target information more efficiently and to very quickly abort further processing of the familiar but non-target events. Older adults, on the other hand, seem less able to differentially allocate attention on the basis of the target-status of the study words.

ERP Responses (Response-Locked). The averaged response-locked waveforms for younger adults for the source memory task are presented in Figure 14. In this figure, the ERN component was most clearly identifiable, being most negative at each site about 60 ms after the response. In contrast, for older adults (Figure 15), the ERN appeared as

part of a gradual negative deflection beginning about 85 ms before the response and returning to baseline about 160 ms after it. The Pe for the source memory task, seen most easily in the waveforms of the younger adults, appeared as two relatively sharp positive deflections, one at about 170 ms, and a second smaller one at about 340 ms post response.[2] For older adults, the only positive deflection appeared near baseline in the waveform, about 190 ms after the response. Thus, the ERPs for the older adults contained ERN deflections that were less negative and Pe deflections that were less positive relative to those of younger adults (see Figure 16 for a comparison of error-related components across age groups).

As can be seen in Figures 14, 15, and 16, the P300 to the word stimulus was missing from all of the waveforms for both groups. The absence of the P300 is likely due to the fact that the source memory task was more difficult than the flanker task. Because of the greater difficulty, the time between stimulus onset and response was much longer (over 1000 ms) so that the P300 to stimulus onset would have occurred well before the behavioural response, and therefore well before the response-locked ERN and Pe. The absence of a P300 immediately preceding the ERN made it unnecessary to select an

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Deciding where the Pe component peaked in the ERP responses for the source memory task was complicated by the presence of two peaks in many individual waveforms, but a single peak in others. Since participants would have prepared and committed themselves to the response about 100 ms prior to its execution, registration that an error had been made would be expected to occur approximately 300 ms after the "point of no return" (Segalowitz, S., personal communication). This timing is approximately consistent with the 170 ms latency of the highest positive peak following the response. Therefore, when there were two peaks, the first of the two positive peaks was taken to represent the Pe. When only one peak was evident, it was selected.

earlier portion of the baseline, as had been the case for the flanker task. Thus, the amplitude and latencies of these components were determined in relation to a baseline beginning 200 ms prior to the response. This short baseline is typical in ERP paradigms.

In the source memory task, lag errors were the most sensitive to age differences, so they served as a basis for ERN analyses. Because several of the younger adults did not make enough errors to form a reliable ERN, their ERP data were omitted from all analyses of error components in the source memory task. Therefore, data entered in the following analysis were from 9 younger and 16 older participants. The mean response-locked amplitude for the ERN and Pe components of the source memory task for these participants are presented in Table 8.

The Source Memory ERN. The ERN amplitude data for participants with sufficient trials to form a stable ERN (younger = 9; older = 16) were submitted to a 4 x 2 mixed model ANOVA with site (Fz, FCz, Cz or Pz) as the within-group factor and age (older vs. younger) as the between-group factor. In general, the ERN was more negative for the younger (M = -6.18 μV) relative to the older (M = -2.75 μV) adults, $F(1, 23) = 8.43$, $p = .008$, $\eta^2 = .27$. This comparison is presented in Figure 16. There was also a main effect of site, $F(3, 69) = 6.68$, $p = .003$, $\eta^2 = .23$. Pairwise comparisons indicated that ERN amplitude was generally more negative at fronto-central sites Fz (M = -4.75 μV), FCz (M = -5.12 μV) and Cz (M = -4.45 μV) than at Pz (M = -3.54 μV). There was no group by site interaction. Thus, the main effects in this analysis parallel those of the flanker task ERN analysis (see Figure 17).

The Source Memory Pe. The Pe amplitudes were analysed in the same way. However, for the Pe, there was no effect of age group, $F(1, 23) = .78$, n.s. The analysis

revealed a significant effect of site, $F(3, 69) = 15.41$, $p < .001$, $\eta^2 = .40$, which was superceded by a site and group interaction, $F(1, 23) = 7.61$, $p = .002$, $\eta^2 = .25$. Post hoc tests confirmed that for the younger adults, the Pe was largest at frontal sites, Fz (M = 5.63 μV) and FCz (M = 5.86 μV), which did not differ, and declined dramatically in amplitude from centro-posterior sites Cz (M = 4.01 μV) to Pz (M = .73 μV). For the older group, the Pe was largest at fronto-central sites, FCz (M = 3.29 μV) and Cz (M = 3.93 μV), which did not differ, but was smaller at Fz (M = 2.21 μV) and Pz (M = 1.80 μV). Thus, the group effect was not significant for the source memory Pe, and the nature of the interactions differed (see Figure 18). For the younger group, the source memory Pe was frontal, whereas in the flanker task it peaked at Cz. For the older group, the source memory Pe was distinctly smaller at Pz, whereas in the flanker task it was evenly distributed across central sites.

In summary, the behavioural and LP results from correct trials in the source memory task reported here are in line with those usually found in such paradigms. In addition, the age effect seen in the ERN amplitudes associated with flanker errors was present in ERN amplitudes associated with the source memory task, suggesting that the error-related processing reflected by the ERNs in the two tasks was similar, irrespective of whether the task involved was primarily perceptual or conceptual. However, the age effect present in Pe amplitudes from the flanker task was absent from Pe amplitudes in the source memory task.

Comparisons across tasks: ERP responses

The next step was to determine whether the error-related ERP components from the flanker task and the source memory task were comparable. In each case, the

respective ERP components of interest were subjected to an analysis of variance followed by appropriate post-hoc comparisons.

ERN. To test whether the ERNs from the source memory task and the flanker task were comparable, the ERN amplitude data from both tasks were submitted to a 2 x 4 x 2 mixed model ANOVA with paradigm (flanker vs. source memory) and site (Fz, Fcz, Cz, Pz) as within-group factors and age (older vs. younger) as the between groups factor. There was a main effect of age group, $F(1, 23) = 27.54$, $p < .001$, $\eta^2 = .55$, indicating that, as expected, the mean ERNs (averaged across four sites) produced by the older adults were generally shallower ($M = -2.43$ μV) than those produced by the younger group ($M = -7.12$ μV). The effect of paradigm, however, was not significant, $F(1, 23) = .28$, *n.s.* There was no interaction between paradigm and group but there was a main effect of site as well as site by group and site by paradigm interactions. These were superceded, however, by a significant three-way interaction among paradigm, site and group, $F(3, 69) = 10.60$, $p < .001$, $\eta^2 = .32$. The ERN produced during the conceptual task, when averaged across groups and sites, was generally comparable in amplitude to the ERN elicited during the perceptual task, for the younger adults. The younger adults produced deeper ERNs during the flanker task (FCz = -11.57 μV) than during the source memory task (FCz = -6.86 μV), but these latter ERNs generally retained their roughly parabolic shape even in the conceptual task. Older adults produced ERNs of about the same amplitude across the flanker (Cz = -3.09 μV) and source memory tasks (FCz = -3.39 μV) but the maximum shifted slightly from central sites for the flanker (Cz) to more frontal sites for the source memory task, (FCz). In addition, while the shallow *flanker* ERN generated by older adults was the expected shape, in the source memory task, the

shape of the older ERN was distorted, in that it became deepest at frontal sites, indicating more frontal activation. Thus, the ERN appeared to reflect the processing demands of the task, for older adults. (Refer again to Figure 17. In addition, topographical comparisons of ERN activity across tasks and age groups are depicted in Figures 19-21).

Pe. Pe amplitude data from both tasks were analysed in the same way. As in the analysis of ERN amplitudes, there was an effect of group, $F(1, 23) = 9.87$, $p = .005$, $\eta^2 = .30$, such that the Pe generated by older adults was smaller ($M = 4.04$ μV) than that generated by younger adults ($M = 7.31$ μV). However, in contrast to the ERN, these components did differ in amplitude as a function of paradigm, $F(1, 23) = 12.51$, $p = .002$, $\eta^2 = .35$, such that the mean Pe to errors in the flanker task ($M = 7.91$ μV) was greater in amplitude than in the source memory task ($M = 3.44$ μV). There was no interaction between paradigm and age group. The effect of site was significant, $F(3, 69) = 20.73$, $p < .001$, $\eta^2 = .47$, but was superceded by a three-way interaction among task, paradigm and group, $F(3, 69) = 19.59$, $p < .001$, $\eta^2 = .46$. Younger adults produced larger Pe's during the flanker task ($M = 10.55$ μV) than during the source memory task ($M = 4.08$ μV) which shifted from more posterior sites (especially Cz) in the flanker task to frontal sites (Fz, FCz) in the source memory task. Older adults produced Pe's that shifted from fronto-central sites (Fz, FCz, Cz) in the flanker task to peak at central sites for the source memory task (FCz, Cz). It was as if the groups swapped Pe profiles in the source memory task, as compared to the flanker task Pe, although the overall age difference in amplitudes remained. Refer again to Figure 18. Topographical comparisons of Pe activity are presented in Figures 22-24. The pronounced shift in the locus of activity for the younger adults from a clearly central-posterior focal point in the flanker task to the very

frontal focus in the source memory task is clearly evident (Figure 24). These foci in younger adults are consistent with the respective natures of the paradigms: the more perceptual task might be expected to elicit an ERP response from centro-parietal regions, and the more difficult task requiring identification of the source of a remembered stimulus might be expected from frontal regions).

Thus, in contrast to the ERN, the Pe did differ as a function of task such that it did not achieve the same amplitude in the source memory task as it did in the flanker task. If the Pe represents conscious registration of an error, as has been suggested by Davies et al. (2001), the source memory task Pe may be smaller because in that paradigm it is more difficult to be certain that an error has been made.

P300/LP. To determine whether the P300 and LP components similarly reflect stimulus evaluation in their respective tasks, the P300 amplitude data from response-locked error trials[3] and LP amplitude from stimulus-locked correct trials were submitted to a 2 x 4 x 2 model ANOVA with paradigm (flanker vs. source memory) and site (Fz, FCz, Cz, or Pz) as within-group factors and age (older vs. younger) as the between-groups factor. The overall amplitudes of these two responses were comparable across age groups, $F(1, 30) = .02$, n.s. The effect of paradigm, however, was significant, $F(1, 30) = 13.74$, $p = .001$, $\eta^2 = .31$, such that the amplitude of the flanker P300 ($M = 4.23$ μV) was larger than the LP response to lag words ($M = 1.29$ μV) in the source memory paradigm. There was a main effect of site, $F(3, 90) = 7.54$, $p = .003$, $\eta^2 = .20$, such that overall, the amplitudes of these two components were larger at more posterior sites (Cz and Pz) than

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[3] The P300 to errors and the P300 to correct trials in the flanker task were not significantly different.

at frontal sites. The interaction between site and group was also significant, $F(3, 90) = 14.27$, $p < .001$, $\eta^2 = .32$, such that for young adults, amplitudes were greater at more posterior sites, but for older adults, amplitudes were greater at fronto-central sites. The interaction between site and paradigm did not reach significance, $F(3, 90) = 2.71$, $p = .10$, $\eta^2 = .08$, nor did the site differences across group interact with paradigm. These data are presented graphically in Figure 25. A topographical comparison of the response-locked P300 in the flanker task to error trials for each age group is provided in Figure 26. The obvious lateral and anterior-posterior differences across age groups bear further investigation, but such work is beyond the scope of this thesis.

The results of this analysis reflect an important difference in the psychological import of these positive ERPs: a large P300 to a stimulus is desirable because it reflects the allocation of attention to the stimulus (e.g. Van der Stelt et al., 1998). However, a large LP to a lag word in the source memory task is inappropriate, since as non-target information, lag words presumably should be ignored.

Cardiac Responses

Parasympathetic control of heart rate was assessed to determine its association with behavioural and electrophysiological indices of error processing in the perceptual (flanker) and conceptual (source memory) attentional tasks. Cardiac data may be examined with reference to mean interbeat interval (IBI), its standard deviation (SD), and its coefficient of variation (CV). It can also be examined on the basis of vagal tone (VT), which is based on respiratory sinus arrhythmia (RSA). VT provides information about the size of the small, vagally-mediated changes in heart rate that are related to respiration, and can be considered an estimation of vagal function. Full analyses were undertaken for

each of these indices of cardiac function. It was clear that, as predicted, VT was most sensitive to group and task differences and will thus be the focus of this section. Nonetheless, for those interested, analyses of IBI and its standard deviation and coefficient of variation can be found in Appendix C.

Three kinds of VT measures were used in the analyses which follow: VT measured at rest, which could be considered a trait measure of characteristic levels of heart rate variability (HRV), VT level during task performance, which could be considered a state measure of HRV, and a residual variable computed as the variability remaining in on-task VT when the variability inherent in baseline VT has been regressed out. This last measure represents VT variance associated with task, controlled for characteristic baseline levels. By comparing on-task VT to baseline VT, one can estimate the downward group shift from baseline levels that occurs with task performance. Parasympathetic control, as measured by these VT variables, was expected to vary as a function of age group and of task demands.

Three baseline cardiac recordings were made during which the participant was at rest: the first, before any tasks were begun, the second, after the first block of the source memory task but just prior to the flanker task, and the third, after all tasks were completed. These are referred to respectively as Baseline 1, Baseline 2 and Baseline 3 (see Table 9). Cardiac data were also collected during performance of both the flanker and source memory tasks. The flanker task, being lengthy, allowed for three sections of VT data of about three minutes each: early, middle and late, referred to as Flanker 1, Flanker 2 and Flanker 3, respectively. The source memory task was done in 2 blocks

(Block 1 and Block 2). Each of these allowed for 2 sections of VT data (Block 1a and Block 1b; Block 2a and Block 2b).

Baseline Vagal Tone. The data from the three resting vagal tone measures were submitted to a mixed model ANOVA with baseline period (Baseline 1, 2 or 3) as the within-group factor and age (older vs. younger) as the between-group factor. Not surprisingly, there was an effect of age, $F(1, 27) = 31.58$, $p < .001$, $\eta^2 = .54$, indicating that older adults ($M = 4.17$) had characteristically lower levels of resting VT than younger adults ($M = 6.41$). A main effect of baseline period was found, $F(2, 54) = 6.36$, $p = .003$, $\eta^2 = .19$, such that mean vagal tone increased steadily across the three baseline conditions (Baseline 1 $M = 5.1$; Baseline 2 $M = 5.2$; Baseline 3 $M = 5.6$), reflecting a very gradual increase in heart rate variability during the rest periods across the testing session (see Figure 27). Baseline 1, however, was taken as most representative of participants' resting vagal tone levels, since it was calculated from data collected in the period before any testing was begun. Therefore, Baseline 1 VT was used as the covariate for forming residualized variables and in analyses relating VT to other variables. Hereafter it is referred to simply as baseline VT. There was no interaction with group.

Vagal Tone Associated with the Flanker Task. VT levels derived from cardiac recordings during the flanker task were submitted to a mixed model ANOVA, with flanker period (early, middle, or late) as the within-group factor and age (older vs. younger) as the between group factor. Again, the effect of age was significant, $F(1, 26) = 29.93$, $p < .001$, $\eta^2 = .54$, with older adults ($M = 3.66$) showing lower levels of VT than their younger counterparts ($M = 5.76$), but the effect of flanker period failed to reach significance, $F(2, 52) = 3.14$, $p = .062$, $\eta^2 = .11$. Therefore, VT levels from the three

flanker periods were collapsed for further analyses. There was no interaction between period and group.

To examine on-task VT while controlling for baseline VT, a residual variable was computed whereby Baseline 1 VT was covaried from mean VT during flanker task performance (VTresid). VTresid did not differ between groups, $t(26) = 1.60$, $p = .12$, suggesting that the group shift from baseline VT to task level VT was about the same for both age groups on this task.

Vagal Tone Associated with the Source Memory Task. Likewise, the VT levels derived from cardiac recordings during the two blocks of the source memory test were submitted to a mixed model ANOVA, with block (Block 1, Block 2) and period (early vs. late) as the within-group factors and age (older vs. younger) as the between-group factor. Again, the age effect was significant, $F(1, 25) = 38.4$, $p < .001$, $\eta^2 = .61$, indicating that in this task as well, VT was lower in the older group. This analysis also revealed a main effect of block, $F(1, 25) = 21.71$, $p < .001$, $\eta^2 = .47$, such that VT was higher in the second block ($M = 5.23$) than in the first ($M = 4.69$). The VT rise in Block 2 is likely due to the fact that the task is no longer new to the participants. There was no effect of test period, $F(1,25) = 2.67$, n.s., indicating that VT did not change within each block. Therefore, in any further analyses involving vagal tone levels during the source memory task, vagal tone was collapsed across periods and referred to simply by block. None of the interactions in this analysis was significant.

Residualized VT variables were also computed by regressing the vagal tone associated with baseline (Baseline1) from each block of the source memory task. These were submitted to a 2 x 2 mixed model ANOVA with block as the within-subjects factor

and age as the between-subjects factor. No significant effects were found. Thus, all following correlations between on-task VT in the source memory task and other variables are restricted to Block 1.

Vagal Tone by Task Comparisons. To compare levels of VT across paradigms, mean VT levels from the flanker task and the first block of the source memory test[4] were submitted to a 2 x 2 mixed model ANOVA with paradigm (flanker vs. source memory) as the within-group factor and age (older vs. younger) as the between group factor. VT was higher in the younger group ($M = 5.82$) than in the older group ($M = 3.54$), $F(1, 26) = 33.57$, $p < .001$, $\eta^2 = .56$. Importantly, there was no effect of paradigm, and the interaction of age with paradigm failed to reach significance, $F(1, 26) = 3.27$, $p = .08$, $\eta^2 = .11$. Similarly, VTresid derived for the two paradigms were also submitted to a separate 2 x 2 ANOVA with paradigm (flanker vs. source memory) as the within-group factor and age (older vs. younger) as the between group factor. In this analysis, the age effect for VTresid did not reach significance, $F(1, 26) = 2.96$, n.s., nor was there any effect of paradigm or interaction.

Taken together, the results of these analyses suggest that regardless of baseline levels and regardless of task, both groups shifted about the same amount from baseline to task, with the result that the age difference present in baseline VT was preserved in

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[4] Comparing on-task VT from the first block of the source memory task with VT from the flanker task was considered appropriate because in both cases, what was of interest was the level of VT as individuals began each task. The second block of the source memory task was included in the experiment only to ensure sufficient trials to make ERP averages and represented a second presentation of the same task to the same individuals. Thus, there may have been some adaptation here that would not have been possible in the flanker task, which was presented only once.

absolute levels of VT during task. This indicates that heart rate variability in younger adults was greater, i.e., more influenced by vagal stimulation and withdrawal, both during rest and during task performance.

Relationships Among VT, Behavioural Outcomes and ERP responses

The consistent age gap in vagal tone seen here raises the question as to whether the effects of aging on the cardiovascular system may account statistically for age differences in task performance and ERP responsivity. Thus, it was predicted that individual task performance and also individual ERP responsivity, in terms of ERN and Pe amplitudes, would vary as a function of parasympathetic control. As such, those with greater control of the vagal brake were expected to perform better on both the perceptual and conceptual tasks, and were expected to produce ERP components with larger deflections, positive or negative – a more informative discovery. The speculation was also made that the more attentional control required in a task, the more important the link between VT and performance would be. To test these hypotheses, a series of hierarchical regressions was carried out using behavioural measures as the dependent variable, and age, VT variables or the interactions between age and VT variables as predictors. In each regression, age was entered on the first step, so that any overlapping variance between age and VT variables would be attributed to age. The issues of interest were whether VT variables would add any information to the prediction of the dependent variables beyond that provided by knowledge of age alone, and whether VT related to the dependent variables differently in one group versus the other.

To the extent that ERPs represent the allocation of attention to task-relevant stimuli, it was anticipated that the amplitude of these components would be sensitive to

variations in VT. To examine this issue systematically, a series of hierarchical regressions was performed for each ERP component of interest: the ERN and Pe associated with errors in both tasks and the LP amplitudes associated with processing non-target stimuli in the source memory task. Each of these components served as dependent variables in regression analyses in which age was always entered on the first step, followed by one of the various VT measures (baseline VT levels, VT levels during task, and VTresid) on the second and the appropriate interaction term on the third. The results of these regressions were taken to indicate whether VT provided additional information beyond knowledge of age group in predicting ERP amplitudes. In order to offset the high probability of Type I error in these exploratory analyses, the criteria adopted for a reportable effect were set high using a "protected F" strategy. No VT variable was accepted as a predictor of errors or ERP amplitudes unless the ANOVA assessing the model as a whole, i.e., the model including the most complex term in the regression (the interaction between age group and VT), was significant, in addition to the ANOVA assessing the VT term itself.

Prediction of Flanker Errors. Errors in the flanker task were entered into a regression model in which age was entered on the first step, followed by baseline VT and the interaction of age with baseline VT. None of the variables was found to be a significant predictor of flanker errors. In the next regression model, VT during the flanker task was entered as the second predictor, after age, followed by the interaction term. No predictors were significant. Finally, VTresid was entered on the second step, after age, followed by the interaction term and again, no predictors were significant. These results indicate that neither age nor VT accounted for variance in error production in the flanker task.

Prediction of Source Memory Errors. A second series of regression analyses was performed with source memory errors as the dependent variable. Again, age was entered on the first step, baseline VT on the second step, and their interaction on the third. Age was found to be an excellent predictor of source error on this task, accounting for 47% of the variance, $t(27) = 4.85$, $p < .001$). Baseline VT did not add to the prediction of source error when entered on the second step but the interaction between age and baseline VT approached significance, accounting for an extra 7% of the variance in source memory error, $t(25) = -2.03$, $p = .053$. As can be seen in Figure 28, higher resting VT was marginally associated with fewer source memory errors, but only for the older group. Although there were exceptions, being an older adult usually located a participant at the lower end of the VT scale in comparison to younger adults, and also made it highly likely that he would make numerous errors in the source memory task. According to these data, however, *within* the vagally-disadvantaged older group, the higher one's resting level of VT, the fewer times one would be likely to mistake a lag word for a study word – for an older adult.

Using the same procedure, VT level during the first block of the lag task (Block 1) was not found to predict source memory error. Similarly, residual VT from Block 1 also failed to add to the prediction of source errors. Neither accounted for a significant amount of variance over that predicted by age. The interaction terms were not significant in these analyses.

In summary, after the considerable effectiveness of age in predicting source memory errors had been taken into account, baseline VT added marginally to the prediction of source error, but only for the older adults. Therefore, source errors in this

study were partly contingent on VT measures, for this group. That is, those older adults with better vagal control over heart rate variability at rest were more likely to make fewer errors in a psychologically demanding task. Those older adults with higher VT at rest were in that respect more like younger adults, whose VT is characteristically high. This is consistent with their making fewer errors. However, the relationship was not true for the younger adults in the source memory task and did not hold for either group in predicting the more perceptual errors on the flanker task.

Prediction of ERN amplitude in the Flanker Task. In the first regression analysis involving ERPs, the dependent variable was the amplitude of the ERN in the flanker task. Age was entered on the first step, baseline VT on the second step and their interaction, on the third. Only age group proved to be a significant predictor of ERN amplitude, $t(27) = 2.79$, $p = .01$, accounting for 22 % of ERN variance. The results were the same when the level of VT associated with task performance was tested, either as an absolute level or as a shift from baseline (VTresid). In every case, only age was a significant predictor. Thus, it was concluded that VT did not add any information beyond age in predicting ERN amplitude in the flanker task.

Prediction of Pe Amplitude in the Flanker Task. In the same way, Pe amplitude was entered as the dependent variable into a hierarchical regression model with age group as the first predictor, baseline VT as the second, and the interaction, third. Only age predicted Pe amplitude, $t(27) = -3.41$, $p = .002$, accounting for 30% of the variance. In a fourth analysis, mean on-task VT was entered on the second step, after age. Again, age alone proved to be significant. However, when VTresid was added after age, it accounted for an additional 13% of the variance, $t(25) = 2.30$, $p = .03$. This indicates that the

smaller the residual VT (i.e., less negative; the less VT changed from baseline to test) the more positive (larger) were the amplitudes of the Pe. The interaction term was not significant, indicating that this relationship was similar in both age groups (see Figure 29).

Thus, for the flanker task, younger adults produced error-related ERP components of greater amplitude than did older adults. Levels of VT at baseline or during task did not account for added variance in the prediction of ERP amplitudes. However, the relationship between VTresid and the amplitude of Pe indicates that those who declined less from baseline to test were more likely to produce a Pe of greater amplitude than those who changed a great deal from baseline to test. This may indicate that those who could afford to remain relatively relaxed during the speeded task (i.e. reduce their VT less) were better able to detect their errors.

Prediction of LP Amplitude in the Source Memory Task. The LP component examined here was associated with targeted study words, i.e., those words that were from the study list. Ideally, one should be able to abort extensive processing of the non-target, repeated lag words, which would result in a low LP amplitude to them. A high amplitude to these non-target words suggests that the individual is not able to withdraw attention from information irrelevant to the task. The higher the amplitude of the LP to study words with respect to lag words, the more efficient and flexible the attentional allocation during the task. To test the hypothesized relationship between VT and ERP responses to both kinds of words, it was preferable to use a residual LP measure that represented "targetness". Accordingly, the LP to lag words was regressed out of the LP to study

words to form a new variable, LPresid, thus isolating the ERP response to targeted study words.

Regression models were constructed with LPresid amplitude as the dependent variable. As predictors, age was entered on the first step, a VT variable on the second step, and the appropriate interaction term on the third. Successive regression models employed the baseline VT measure, the VT level during the onset of the source memory task (Block 1), and VTresid (Block 1). By the criteria set for these regression analyses, none of the VT variables predicted the source memory target effect.

Prediction of ERN Amplitude in the Source Memory Task. ERN amplitude from the source memory task was entered as the dependent variable in the next series of hierarchical regression models with age group entered on the first step, a VT variable on the second step, and the relevant interaction term on the third. As in the flanker task, where age was a consistently significant predictor of ERN amplitude, in the source memory task, age again predicted ERN amplitude, accounting for 25% of its variance. Resting baseline VT accounted for an additional 26% of the variance in the amplitude of the ERN, $t(20) = 3.29$, $p = .004$ (see Figure 30). Contrary to predictions, these results indicated that lower resting vagal tone predicted *more* negative ERN amplitudes for older and younger adults.

Mean on-task VT (Block 1) entered after age in this model did not add to the prediction of ERN amplitude nor interact with age. However, when VTresid from Block 1 was entered on the second step of a regression model, the residual itself accounted for 18% of the variance in ERN amplitude, $t(19) = -2.41$, $p = .03$, after age (see Figure 31). Smaller VT residual levels (i.e., less change from baseline to task) was associated with

larger ERNs, for both younger and older adults. Generally speaking, although ERN amplitudes for older adults are typically shallower than those of young adults, in this study they seemed equally contingent on VT measures.

Thus, it would appear that low baseline VT was associated with larger ERNs during the source memory task. Additionally, smaller vagal tone residuals, representing smaller shifts in vagal tone from baseline to task, were associated with larger ERNs. Both low vagal tone and small shifts in vagal tone may indicate some inflexibility in parasympathetic control of heart rate. It would seem that when engaged in monitoring the source of item information, the less parasympathetic mediation of the heart, the greater the ERP reaction to having made an error. Alternatively, since lower VT influence and increased errors are also associated, higher numbers of errors could be an intermediate variable between VT and ERN amplitudes.

Prediction of Pe Amplitude in the Source Memory Task. Following the same strategy as above, hierarchical regressions were carried out to predict Pe amplitude in the source memory task from VT variables. In these analyses, age was never a significant predictor of Pe amplitude, despite being the earliest entry. In the first regression model, however, baseline VT was found to be an important predictor of Pe amplitude when entered on the second step, $t(20) = 3.79$, $p = .001$, accounting for 40% of the variance (Figure 32). The interaction term added no new information to the prediction. Thus, the higher the resting vagal tone, the more positive the Pe, for both groups. Similarly, mean on-task VT from Block 1 was entered as the VT predictor in the next hierarchical regression model. On-task VT was not found to be a significant predictor of source

memory Pe amplitude, nor was there any significant relationship between VTresid variables and the Pe in the source memory task.

The results of these analyses would suggest that the greater the activity of the parasympathetic system at rest, the larger the Pe amplitude to source errors. However, it was resting VT prior to task onset that accounted for most of the variance in the Pe (40%) after age. In fact, higher VT predicted a more positive waveform overall, that is, a less negative ERN and more positive Pe.

This raises the possibility of a confound in the data. As was seen in the stimulus-locked waveforms to correct source memory trials in Figure 13, the waveforms of younger adults depicted an obvious 'dip' around the time of the response, especially at more posterior sites. This negative deflection varied by word type, being larger for study and lag words than for foils. Since the ERN occurred within 100 ms of the response, such an aberration in the waveform could have systematically altered the baseline with respect to which the ERN and Pe amplitudes were calculated. In order to assess whether the relationships between VT variables and these ERPs were confounded with a possible baseline shift, the main ANOVAs and regression analyses were repeated using ERN and Pe amplitudes derived from the source memory difference wave (see Figure 33; statistics for the ANOVAs not reported here). Using the difference wave, i.e., subtracting the waveform to correct trials from the waveform to incorrect trials and re-scoring the ERN and Pe, eliminates the possibility that a baseline shift on correct trials is confounding the relationship between VT and the error-related ERPs. When this was done, the predictive relationship between baseline VT and the difference wave ERN was still found to be significant, although it was no longer "protected" as the average wave ERN had been, but

no other relationships between VT variables and the amplitude of either the difference wave ERN or Pe were significant. Correlations between baseline VT and the difference wave ERPs were also not significant, although their direction was the same as for correlations involving the average wave ERPs (positive). Thus, although contrary to original predictions, it appears unlikely that the positive relationship of baseline VT to ERN amplitude found in these analyses was due to a confound with the negative 'dip' in correct trials during the baseline period for younger adults. It is clear that explicating the nature of such relationships will require additional research.

Summary

The relationships among age, VT variables and the outcome variables (errors and ERPs) in the two tasks may be summarized in the following ways. For the flanker task, neither age nor VT variables predicted errors. Moreover, while age was a significant predictor of flanker ERN amplitude, none of the VT variables accounted for any variance beyond age in predicting ERN amplitude in this task. The situation was similar for the flanker Pe. Age significantly predicted Pe amplitude in all of the regression analyses, whereas VT variables did not – with one exception. Residual VT was correlated with Pe amplitude, such that smaller shifts were associated with larger Pe's. Overall, though, VT variables were poorly linked to either errors or ERP amplitudes in the flanker task.

The source memory task is known to be particularly sensitive to age-related changes in attentional control. Not surprisingly, age group was an excellent predictor of errors in the regression analyses that were carried out. Beyond age, however, baseline VT added marginally to the prediction of errors but only for older adults, with lower levels of baseline VT related to higher numbers of errors. Thus, for older adults, low levels of

parasympathetic control were not conducive to accurate source-memory discrimination on this task.

There were also significant relationships between VT and the error-related ERP components in the source memory task. The amplitude of the ERN was best predicted by baseline and residual levels of VT. The lower the baseline VT, or the smaller the residual, the greater the amplitude of the ERN. This suggests that poorer vagal influence leads to a greater ERP response to having made an error on this task. Moreover, this finding was true for both age groups, even though the ERNs of the older group were relatively attenuated. In addition, larger Pe's were clearly associated with higher VT in both groups. Like the ERN, the Pe appears to be influenced by parasympathetic effects on cardiac response.

Discussion

The issue of what allows humans to control the allocation of attention has occupied psychologists for some time. Although many factors might be examined in this pursuit, ERPs generated during the performance of cognitive tasks provide precise and immediate measures of the outcome of attentional processes and can be used to track how attentional allocation differs with age, and by individual. In the present study, these differences were investigated in the context of error-processing.

Older and younger adults participated in two contrasting tasks, the successful completion of which required considerable control over attention, although each had different attentional requirements. To date, the tasks used extensively in the study of the ERN have been mainly simple exercises requiring speeded responses, such as the flanker task (Eriksen & Eriksen, 1974). Those studies which have included older participants have reported that the ERNs of older adults were substantially smaller relative to younger adults, but have generally not been informative about attentional control across age groups in cognitively demanding situations. Some researchers contend that age-related differences in error-processing may in fact be responsible for the behavioural differences seen in older adults, such as increased numbers of errors and longer response times (e.g. Falkenstein et al., 2000).

Older adults are known to experience difficulties remembering the source of a memory. Rather than "memory loss" or encoding problems *per se*, deficits in attentional control may provide a more precise explanation for the increased source errors seen among older adults (Dywan et al., 2002). The source memory task was selected for comparison to a response time task because it has proven sensitive to age differences in

attentional control (e.g. Dywan et al., 1998, 2001, 2002). Therefore, ERNs were collected from older and younger adults in the source memory task and compared to standard ERNs elicited by a traditional flanker task. This cross-sectional experiment thus contained two age groups and two levels of task difficulty wherein each group could serve as its own control across tasks. The first major issue examined was whether the ERN from such a conceptual task was comparable to that generated in a simpler, primarily perceptual task.

Evaluating error trials in the traditional source memory paradigm presents some difficulty. Typically, younger adults make relatively few errors in this task, so there are few data against which to compare the performance of older adults. The positive waveform associated with study, lag or foil words is thought to be an ERP response to the salience of a given stimulus (Dywan et al., 2002). Since it is normally calculated in relation to stimulus onset and occurs before a response has been selected, it does not, however, reveal any information about error-processing. The late positivities (LPs) were examined in order to be sure that the results of this experiment compared with typical LP components from other studies in the literature.

However, error-processing in the source memory task could be explored in the context of error-related ERPs, the ERN and Pe. For these components a stable waveform may be produced using fewer trials. Moreover, if the ERPs from both tasks were similar, comparing source memory ERNs and Pe's to those elicited by the flanker paradigm would extend our understanding of error-processing from response time tasks to a more complex task that appears to invoke a different kind of attentional control.

The second major issue addressed by the present research was whether the ability to control the allocation of attention, as evidenced by ERPs, could be predicted by other indices of general physiological reactivity. Vagal tone (VT) is a computed measure of parasympathetic control of heart rate and one of the contributing neural sources to variability in heart rate. A literature review indicated that cardiac function is generally sensitive to changes in attentional requirements and that the parasympathetic system has a unique role in slowing the heart in order to support the voluntary direction of attention in non-threatening circumstances. In addition, previous research from our lab had identified a relationship between VT and ERP responses in a source memory task (Veenhof, 1997), as had the preliminary analysis of another data set (Mathewson, Dywan, Segalowitz et al., 2001).

In the flanker task, participants were asked to identify the central letter in a five-letter array by choosing one of two keys. Hundreds of trials were involved in this task and the presentation rate was fairly quick (SOA = 1250 ms). Thus, the errors made in this paradigm consisted mainly of impulsive responses or action slips. In the source memory task, words were presented to participants for study, after which a source recognition test was given. Test items consisted of new words (foils) and words that had been seen previously, either in the study list, or as repeated words in the recognition test (lag words). The source memory task required participants to discriminate between the familiarity associated with repeated, non-target words and that associated with targeted words from the study list. Participants were asked to respond "yes" when they saw a word from the study list, and "no" to all other words.

Behavioural Outcomes. The behavioural outcomes of both tasks constituted replications of the findings typical of their respective paradigms. In the letter-based flanker task, older adults tended to make more errors and were slower to respond than their younger counterparts. Both groups, however, made more errors and were slower to respond to trials in which the flanking letters were incongruent. Younger adults were proportionally faster on congruent trials than incongruent trials than were older adults.

In the source memory task, older adults had a greater tendency to say "yes" to test items overall, and responded more slowly than did the younger group. They also made three times as many source errors (49%) as did younger adults. That is, they were three times as likely as the younger adults to categorize familiar but non-target words as having been in the study list. The high error rate occurred despite older adults' showing other indicators of good performance. For instance, the groups did not differ in their identification of study words or in their rejection of non-familiar foils. Therefore, these findings replicate previous reports of increased numbers of source memory errors in older adults (e.g., Dywan, et al., 1998, 2001, 2002; Veenhof, 1997).

Electrophysiological Outcomes. The ERP results of the flanker task may also be regarded as a replication of earlier studies comparing the cortical responses of older and younger adults. The ERN component could be predicted on the basis of age, being shallower for older relative to younger adults. The ERN generated by the younger adults appeared as a pronounced negative deflection, maximal at FCz, as is typically seen in flanker tasks. It did not differ across congruency conditions. In older adults, the ERN was more diffuse. The Pe component was also quite flat in the waveforms of the older adults. However, the P300 component representing registration of the stimulus was the

same for both age groups. Without the negative finding regarding the P300, it might be concluded that smaller ERPs were the inevitable output of an older brain. Such is not the case, however, and an explanation for the shallow error-related components must be sought elsewhere. Correlational analyses indicated that latency jitter also be ruled out as the cause of the age-attenuated waveforms.

Before examining ERNs to source errors and to ensure that the present results were generally comparable to those found in other source monitoring research, averaged waveforms were stimulus-locked in the usual manner, and the late positivity (LP) from these participants examined. As is typically found in source memory tasks, older adults generated a large LP to repeated lag words, whereas the waveforms of younger adults discriminated clearly between study words, lags and foils.

In earlier reports, the large LP to the repeated lag words on the part of older adults has been interpreted as an "inappropriate" ERP response to non-target information. (It also refutes the idea that older adults are subject to a general decline in overall EEG power). The presence of large LPs to lag words on correct trials suggests that even when older adults have successfully categorized lag words as non-study words, they have been unable to inhibit a large positive electrophysiological response to them. As such, older adults appear to have made their lag-item categorizations on the basis of late correction – having seen the word, they responded electrophysiologically as if it were a target, then overcame that reaction to categorize the item successfully. Younger adults, who typically do not produce an inappropriate ERP response to lag words, appear to have been able to categorize such words on a basis of early selection. It is as if they knew immediately that the repeated, lag words were non-target information–without producing any large,

electrophysiological response to them. They were able to abort any further attention to these words, making correct categorizations at once (Dywan, et al., 2002).

Since the results from correct trials in the present study replicate those reported in the literature, it was deemed reasonable to also examine error trials from this task and to compare them with error trials from the flanker task. For examination of the ERN and Pe components, the waveforms were re-averaged, locked to the response. In the source memory paradigm, ERNs of older adults were shallower than those of the younger adults, the same way they were in the flanker task. Thus, the significant age effect seen in the flanker ERN was retained in the source task ERN. Visual inspection of the ERN components of younger adults from the two tasks suggested that, although they had the same general shape, the source memory ERN might be smaller than the ERN to flanker errors. However, a cross-task analysis of the mean ERNs in the two tasks revealed no difference in ERN amplitudes for younger adults. Given these results, it seems reasonable to conclude that for this group at least, the ERN generated in response to errors in a conceptual task was generally comparable to that elicited by simpler response time tasks. However, the site of greatest activation in the source memory ERN for older adults was more frontal than for the ERN to flanker errors. Thus, the ERN of older adults seemed to reflect the processing demands of the task. Similar results were reported by Dehaene et al. (1994) with respect to an error-monitoring task involving semantic categorizations. Using BESA (Scherg & Berg, 1995), Dehaene and colleagues identified a dipole for this task which suggested that its generator's most probable location was more anterior than a dipole calculated from a simple speeded response task. Our conclusions should be accepted with caution, though, as topographical maps and peak analyses

provide information about electrical activity as it is picked up at the scalp, but not the specific locations of the neural generators responsible for it. The findings reported here should therefore be verified by submitting these ERP data to an analysis program capable of dipole localization, such as BESA. If the ERN in the conceptual task is comparable to that from the perceptual task in younger adults, one would expect its generator to be located in about the same region of the ACC, as the flanker ERN. This question will be addressed in subsequent analyses but is beyond the scope of this thesis.

Evidence for the Pe as awareness of error. Although the focus of this study was the investigation of the ERN component in error-processing, the Pe results seemed interesting themselves and worthy of follow-up. The Pe component from the present flanker task appeared to be somewhat more sensitive to changing conditions than the ERN. For example, in subsidiary analyses it was observed that the Pe was sensitive to congruency condition, being larger for congruent than incongruent trials, whereas the ERN was not. In such trials it was also easier to be certain of having made an error, where all the letters matched, than in incongruent trials. In addition, the Pe was larger in the perceptual task. Since flanker stimuli were simple and responses were made immediately after stimulus presentation, it was easier to be sure when an error was made in that task. Conversely, it was comparatively difficult to know whether one had made an error in the source memory task, given the time span between study and test and the difficulty involved in determining where a word was last seen. Thus, two observations from this study support the notion that the Pe represents awareness of having made an error. In both cases, its amplitude distinguished between differing error types, being larger for the errors that were easier to detect.

Overall Pe amplitude – averaged across sites – did not differ by age in the source memory task. However, the amplitude of the Pe did differ across groups on the basis of task and site. For younger adults, the site of maximal Pe activation changed radically, depending on the task under consideration. For this group, although the *flanker* Pe appeared to be generated over central sites, the source memory Pe was clearly largest at very frontal sites. The profound change in scalp topography of the Pe, in younger adults at least, appeared to reflect the nature of the demands posed by the task, since it was produced parietally in a perceptual task, and frontally in a task requiring executive functions, i.e., judgments about the source of a recent memory. No such relationship was evident in the topography of the source memory Pe for older adults.

Taken together, these results suggest some degree of independence between the Pe and the ERN. The ERN may differ from the Pe in not being *directly* related to conscious awareness of having made an error. For example, the ERN has been shown to occur in adults who are unaware that they have made an error, in a task involving saccadic eye movements (Nieuwenhuis, Ridderinkhof, Blom, et al., 2002). Although task instructions or the addition of incentives can influence average ERN amplitude from the point of view of an overall mind-set (Gehring et al, 1993; Pailing et al., in preparation) – and perhaps also Pe amplitudes– it seems that the ERN is somewhat automatic and that conscious detection of an individual error is reflected in a component other than the ERN itself.

Why is the older ERN smaller? Older adults are typically very conscientious research participants. For the most part, the older participants in this study appeared to be aware of the errors they made, since most of them showed obvious signs of exasperation

at the time. Based on their conscientiousness, older adults would be expected to generate larger ERNs rather than smaller ones. Experimental evidence indicates that when participants strive for accuracy over speed, ERN amplitudes are larger and that error-monitoring processes are enhanced in people with obsessive compulsive disorder, (Gehring et al., 2000; Gehring et al., 1993).

There may be a simple explanation for diminished ERNs in older adults. Several studies indicate that ERN amplitude appears to depend partly on awareness of having made an error (Luu, Flaisch & Tucker, 2000). For example, when errors are difficult to detect (Falkenstein et al., 1996) or when they have been committed "honestly", i.e. a person does not realize that her choices happen to be wrong, ERNs are smaller (Scheffers & Coles, 2000). If awareness is necessary for ERN production, then on those trials where they made honest errors, older adults would not generate an ERN. If these trials were then averaged in with other trials on which older participants were aware of having made errors, the resulting waveform could be significantly flattened. The attenuated ERN averages of older adults found here might therefore be attributable to older adults' being uninformed about some of their errors. One way to deal with this would be to go back through the data, selecting trial by trial only those error trials in which an ERN was present for inclusion in the averages. However, in a recent study where this was carried out, analyses of single trials from older participants confirmed that the ERN amplitudes in their waveforms were indeed reduced as compared to younger adults (Falkenstein, Hoorman and Hohnsbein, 2001). Thus, the finding that the ERNs of older participants were smaller rather than larger than those of younger adults appears to be a genuine phenomenon requiring an alternative explanation.

According to the theory put forth by Holroyd and Coles (2002; see also Nieuwenhuis, Ridderinkhof, Talsma et al., 2002), when the consequences of an action are suddenly worse than expected, as would be the case when one makes an error, the mesencephalic dopamine system reduces its output. This phasic drop in dopamine acts as a negative reinforcement signal to the basal ganglia and the ACC. The ACC responds to the signal by delegating one of a number of neural command centres to take control of the motor system in order to deal with the unexpected negative consequences. It may be that the burst of negativity that produces the ERN reflects a kind of "emergency response" to errors, that is, the ACC's marshalling of a neural command centre which will direct the motor system.

If D2 (dopamine) receptors in the ACC are reduced in later life, the net result may be ineffective detection of phasic reductions in available dopamine. That is to say, if older adults have chronically reduced levels of dopamine, they might be less sensitive to subtle shifts in its availability. In that case, being unable to pick up a signal that consequences are worse than expected would be expected to affect subsequent neural events. For instance, any disinhibition in the ventral bank of the ACC would be very weak as a result. Lower numbers of D2 receptors could thus indirectly account for the low amplitudes seen in the ERN components of older adults. Moreover, if a smaller ERN means that for older adults the ACC might be less able to effectively muster a neural command centre when necessary, one would expect to see this reflected in behaviour. Holroyd and Coles (2002) have claimed that the rate of learning is slowed when dopamine output does not effectively signal when consequences are worse than expected, and that *that* is the primary cause of age-related changes in error processing. Evidence

from the present study supports this view. Older adults generated more source errors, as well as smaller ERNs. The above account might also explain low-amplitude Pe's. If older participants could not receive proper error signals regarding their mistakes, they might also be less certain of when they have made errors.

Physiological Adaptability and Modulation. A final goal for the present study was to test whether parasympathetic control of heart rate, as measured by VT, would index behavioural and ERP responses in two tasks. To begin with, heart rate variability, including VT, is known to be reduced in later life (McDonald, 1980; Taylor, Hayano & Seals, 1995). VT measures collected for this thesis generally differentiated between groups, across difficulty levels within a task, and among individuals. Overall, on-task VT did not vary by paradigm. However, group differences in VT were consistent in resting baseline levels and maintained during performance of both tasks. Interestingly, the group differences did not hold for residual VT measures for either the flanker task or the first part of the source memory task. This would suggest that at least at the beginning of a psychological test, the adjustment in VT from baseline to task was the same for both groups.

Cardiac control has been documented as an important factor in situations involving greater mental challenges. In a study investigating the effects of mental stress on sympathetic and parasympathetic control of heart rate, Berntson et al. (1994) discovered that although the *group* pattern of activation under mental stress was reciprocal (parasympathetic activity decreased as sympathetic activity increased), idiosyncratic patterns of parasympathetic and sympathetic activation did not necessarily behave in an equal and opposite manner. Furthermore, such individual patterns of

activation were shown to remain stable across three different psychological challenges, including preparing an impromptu speech, performing mental arithmetic and participating in a response time test. Thus, it appears that individual differences in cardiac control may be specific and reliable.

Regression analyses were carried out to test the hypothesis that VT added unique variance to the prediction of behavioural performance and ERP amplitudes for both tasks beyond age group itself. In every regression analysis, the VT variable was evaluated as a predictor after the contribution of age had been taken into account. The results differed by component and by task. None of the VT variables predicted flanker errors, nor ERN amplitude in the flanker task. The flanker Pe was predictable only by the residual representing on-task VT, controlled for baseline, i.e., the degree to which VT shifted from baseline to task. Thus, on the basis of the flanker task alone, one might conclude that cardiac function bore little relation to errors or ERP amplitudes. Performance in a flanker paradigm is data-driven and tightly time-limited, which might necessitate a relatively high level of sympathetic activity. However, since the ERN effect across task interacted with group and site, it may be that the no relationship could be found between flanker ERN amplitudes and vagal tone because the ERN measure used in the analyses was averaged across four sites, thus missing the specificity of the effect.

In contrast to the flanker task, VT did relate to errors on the source-monitoring task, albeit marginally. Older individuals with higher baseline levels of VT were more able to discriminate the source of familiar information, which is exactly what one would have predicted. The relationships between VT and errors were seen in the task that was cognitively more challenging and very sensitive to age-related differences in attentional

control, but not in the simpler response time task. Taken together with the lack of relationship between flanker errors and VT, these results suggest that the greater the need to control attention to do a task, the more influence VT has.

Residual heart rate variability measures seems to represent shifts in VT from baseline level to task level. The present data suggest that making larger parasympathetic adjustments from a given baseline level in order to deal with a psychological challenge results in more appropriate ERPs responses to targets. In another study requiring memorization of a list (but not identification of the source of the items), Jennings et al. (1990) reported that in terms of making cardiovascular adjustments, older adults speeded up HR in order to support memory processes, although younger adults did not. In the present study, both age groups responded with reduced VT (increased heart rates) during performance of the source memory task, possibly because source memory identification may require more resources than memorization. Individual differences in how much adjustment needs to be made may be informative as well.

Individual differences in resting baseline VT were critical in the present study. The relationship originally hypothesized between VT and ERN amplitude was correct only at a group level. Age was associated with lower VT and smaller ERNs. Hierarchical regression analyses indicated, however, that at the level of the individual, the relationship was in the opposite direction. Within the groups, higher individual levels of baseline VT were associated with shallower ERNs and accounted for substantial amounts of ERN variance. It would appear that for individuals, the direction of my original hypothesis was incorrect. The situation is not as simple as originally theorized, i.e., that a healthy nervous system will produce larger ERP components. But the reality is more interesting:

notwithstanding the effects of membership in a group whose VT is characteristically low, individual VT levels provided predictive information about how large an ERP response would be made to errors. The present data also indicated that after the effects of age had been accounted for, higher levels of baseline VT were marginally associated with fewer source errors in the older group.

For the source Pe, higher individual baseline VT levels consistently predicted larger Pe amplitudes, accounting for 40% of their variance, whereas age bore no relation to the size of Pe amplitudes. Therefore, it is tempting to conclude that higher resting levels of VT, i.e., a characteristically more flexible heart rate, allowed for greater cognisance of errors.

Future investigations. The regression analyses presented here are a preliminary step in the investigation of possible relationships between VT and behavioural and ERP outcomes. In the present analyses, amplitudes for each component were averaged across four midline sites. A more thorough examination would include repeating the analyses for the sites most relevant to each component, for instance, FCz and Cz in the case of the ERN, and Fz, FCz and Cz in the case of the Pe.

In addition, in order to fully assess the effects of complex psychological tasks on cardiac control, a measure of sympathetic control should be incorporated in future studies. Berntson et al. (1994) and others have recommended the pre-ejection period measure for this purpose. Although the influence of parasympathetic control on the heart is about three times greater than that of sympathetic control under resting conditions (Berntson et al., 1994), since parasympathetic and sympathetic control can vary under conditions of challenge with respect to each other in a reciprocal, independent, or even co-activated

manner, a more compete understanding of the relative roles of parasympathetic and sympathetic activity would be gained by the inclusion of such a measure. The case for VT as a predictor would be strengthened if it was shown to be significant and sympathetic influences could be definitely ruled out.

Finally, it would be interesting to investigate the Pe further, since it was sensitive to a variety of parameters, including congruency condition, task differences and individual VT levels in this study. The Pe may turn out to be as informative as the ERN in shedding light on the cognitive processes engaged when errors are made.

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Tables and Figures

Table 1.
Means and Standard Deviations for Proportions of Congruent, Incongruent, and Total Errors in the Flanker Task.

	Error Type		
	Incongruent M (SD)	*Congruent M (SD)*	*Total Errors M (SD)*
Group			
Younger (N = 15)	.09 (.06)	.04 (.04)	.07 (.05)
Older (N = 14)	.12 (.04)	.08 (.05)	.11 (.04)
Total (N = 29)	.10 (.05)	.06 (.05)	.09 (.05)

Note. Response data from two older participants were not reported due to randomness in their responses. Response data were not available for one younger participant because of technical problems. Because of the sizable number of missed trials, error percentages have been calculated as errors/(errors + corrects).

Table 2.
Means and Standard Deviations for Response Times for Congruent and Incongruent Errors and Correct Trials in the Flanker Task.

	Stimulus Type			
	Errors (ms)		*Correct Trials (ms)*	
	Incongruent M (SD)	*Congruent M (SD)*	*Incongruent M (SD)*	*Congruent M (SD)*
Group				
Younger (N = 15)	407 (30)	369 (114)	507 (40)	473 (40)
Older (N = 14)	462 (37)	479 (77)	558 (56)	538 (54)

Note. ResponseTime data from two older participants were not reported due to randomness in their responses. Response Time data were not available for one younger participant because of technical problems.

Table 3.
Amplitudes of Response-Locked Average Wave ERP Components (P3, ERN, Pe) by Electrode Site in the Flanker Task.

		ERP Component		
		P3 *μV*	*ERN* *μV*	*Pe* *μV*
Group	*Site*			
Younger (N = 9)	Fz	2.73	-8.38	7.11
	Fcz	4.83	-11.57	10.82
	Cz	7.23	-9.96	13.27
	Pz	8.62	-2.34	10.99
	M	5.86	-8.06	10.55
	(SD)	(2.60)	(4.03)	(2.55)
Older (N = 16)	Fz	3.59	-1.55	5.53
	Fcz	3.84	-2.57	5.84
	Cz	3.53	-3.09	5.41
	Pz	3.30	-1.24	4.27
	M	3.57	-2.11	5.26
	(SD)	(0.22)	(0.87)	(0.69)

Note: For the younger adults, the reported n = 9, in order that cross-task comparisons between the same participants might be made. Results of analyses of flanker components using 9 young adults and 16 young adults were not different.

Table 4.
Amplitudes of Response-Locked Difference Wave ERP Components (ERN, Pe) by Electrode Site in the Flanker Task.

		ERP Component	
		ERN μV	*Pe* μV
Group	*Site*		
Younger (N = 16)	Fz	-11.87	4.69
	Fcz	-15.39	9.16
	Cz	-15.51	11.18
	Pz	-9.73	10.86
	M	-13.13	8.97
	(SD)	(2.82)	(2.99)
Older (N = 16)	Fz	-7.28	2.96
	Fcz	-8.38	3.71
	Cz	-8.54	3.61
	Pz	-5.98	4.28
	M	-7.55	3.64
	(SD)	(1.18)	(.54)

Table 5.
Means and Standard Deviations for Proportions of Items Judged to be Study Words ("Yes" Responses) in the Source Memory Task.

	Stimulus Type		
	Study *M (SD)*	*Lag* *M (SD)*	*Foil* *M (SD)*
Group			
Younger (N = 16)	.63 (.16)	.16 (.12)	.08 (.08)
Older (N = 16)	.56 (.15)	.49 (.21)	.15 (.13)

Table 6.
Means and Standard Deviations for Response Times for Different Stimulus Types in the Source Memory Task (Correct Trials).

	Stimulus Type		
	Study (ms) *M (SD)*	*Lag (ms)* *M (SD)*	*Foil (ms)* *M (SD)*
Group			
Younger (N = 16)	1055 (218)	1037 (349)	1008 (331)
Older (N = 16)	1189 (163)	1317 (318)	1207 (307)

Table 7.
Mean Stimulus-Locked LP Areas Associated with Different Stimulus Types (Study, Lag Foil) and Electrode Sites in the Source Memory Task.

		Stimulus Type		
		Study LP μV	*Lag LP* μV	*Foil LP* μV
Group	*Site*			
Younger (N = 16)	Fz	1.08	-.20	-1.69
	Fcz	1.07	.01	-1.88
	Cz	2.00	.95	-.92
	Pz	3.25	2.36	.96
	M	1.85	.78	-.88
	(SD)	(1.03)	(1.17)	(1.30)
Older (N = 16)	Fz	2.33	2.51	1.69
	Fcz	1.38	1.71	.85
	Cz	.73	1.21	.33
	Pz	1.02	1.76	.78
	M	1.37	1.80	.91
	(SD)	(.70)	(.54)	(.57)

Table 8.
Amplitudes of Response-Locked ERP Components (ERN and Pe) Associated with Lag Words and Different Electrode Sites in the Source Memory Task.

		ERP Component	
		ERN μV	*Pe* μV
Group	*Site*		
Younger (N = 9)	Fz	-6.01	5.63
	Fcz	-6.86	5.86
	Cz	-6.48	4.10
	Pz	-5.37	.73
	M	-6.18	4.08
	(SD)	(.64)	(2.37)
Older (N = 16)	Fz	-3.48	2.21
	Fcz	-3.39	3.29
	Cz	-2.43	3.93
	Pz	-1.72	1.80
	M	-2.75	2.81
	(SD)	(.84)	(.98)

Note. ERP data for participants having $\geq$ 7 error trials are reported here.

Table 9.
Mean Vagal Tone Values by Condition (Baseline 1, Flanker Task, Source Memory Task) and by Age Group.

	Condition			
	Baseline1	*Flanker*	*Source Block1*	*Source Block 2*
Group				
Younger (N = 16)	6.17	5.76	5.89	6.56
Older (N = 16)	4.00	3.66	3.42	3.91

Table 10.
Correlations between Vagal Tone (VT) and other Cardiac Measures (Inter-beat Interval, IBI, Coefficient of Variation, CV, Standard Deviation of Inter-beat Interval, SD) by Condition (Baseline, Flanker, Source Block 1, Source Block 2) controlling for Group.

	Condition			
	Baseline1	*Flanker*	*Source, Block 1*	*Source, Block 2*
	p r	*p r*	*p r*	*p r*
	n=29	*n=28*	*n=29*	*n=28*
Measure				
Mean IBI	.32	.39*	.33	.45*
Mean CV	.70***	.66***	.78***	.67***
Mean SD	.71***	.67***	.76***	.70***

Note. * $p = < .05$; *** $p < .001$. In the baseline condition, baseline 1 VT is correlated with baseline 1 IBI, baseline 1 CV and baseline 1 SD.

Table 11.
Correlations between Inter-beat Interval (IBI) and other Cardiac Measures (Vagal Tone, VT, Coefficient of Variation, CV, Standard Deviation of Inter-beat Interval, SD) by Condition (Baseline, Flanker, Source Block 1, Source Block 2) controlling for Group.

	Condition			
	Baseline	*Flanker*	*Source, Block 1*	*Source, Block 2*
	p r	*p r*	*p r*	*p r*
	n=29	n=28	n=28	n=28
Measure				
Mean VT	.32	.39*	.33	.45*
Mean CV	.16	.14	.11	.16
Mean SD	.45*	.39*	.35	.40*

Note. * $p = < .05$. In the baseline condition, baseline 1 IBI is correlated with baseline 1 VT, baseline 1 CV, and baseline 1 SD.

Table 12.
Correlations between Vagal Tone (VT) and other Cardiac Measures (Inter-beat Interval, IBI, Coefficient of Variation, CV, Standard Deviation of Inter-beat Interval, SD) by Condition (Baseline, Flanker, Source Block 1, Source Block 2) and Group (younger vs. older).

		Condition			
		Baseline	*Flanker*	*Source, Blk 1*	*Source, Blk 2*
		r	*r*	*r*	*r*
Group	*Measure*				
Younger (N = 15)	Mean IBI	.67**	.66**	.54*	.61*
	Mean CV	.83***	.79**	.88***	.90***
	Mean SD	.83***	.86***	.92***	.90***
Older (N = 13)†	Mean IBI	.05	.27	.22	.38
	Mean CV	.54*	.84***	.87***	.36
	Mean SD	.61*	.86***	.86***	.61*

Note. * $p = < .05$; ** $p < .01$; *** $p < .001$. † For the correlations for older adults involving baseline measures, n = 14.
Baseline 1 VT is correlated with baseline 1 IBI, baseline 1 CV and baseline 1 SD.
Mean flanker VT is correlated with mean flanker IBI, mean flanker CV and mean flanker SD.
Mean Block 1 VT is correlated with mean Block 1 IBI, mean Block 1 CV and mean Block 1 SD.
Mean Block 2 VT is correlated with mean Block 2 IBI, mean Block 2 CV and Mean Block 2 SD.

Table 13.
Correlations between Inter-beat Interval (IBI) and other Cardiac Measures (Vagal Tone, VT, Coefficient of Variation, CV, Standard Deviation of Inter-beat Interval, SD) by Condition (Baseline, Flanker, Source Block 1, Source Block 2) and Group (younger vs. older).

		Condition			
		Baseline *r*	*Flanker* *r*	*Source, Blk 1* *r*	*Source, Blk 2* *r*
Group	*Measure*				
Younger (N = 15)	Baseline 1 VT	.67**	.66**	.54*	.61*
	Mean CV	.69*	.30	.23	.46
	Mean SD	.82**	.51	.46	.63*
Older (N = 14)†	Baseline 1 VT	.05	.27	.22	.38
	Mean CV	-.37	-.17	-.16	-.27
	Mean SD	-.08	.41	.22	.32

Note. * $p = < .05$; ** $p < .01$. † For the correlations for older adults involving measures from Block 2, n = 13.
Baseline 1 IBI is correlated with baseline 1 VT, baseline 1 CV and baseline 1 SD.
Mean flanker IBI is correlated with mean flanker VT, mean flanker CV and mean flanker SD.
Mean Block 1 IBI is correlated with mean Block 1 VT, mean Block 1 CV and Mean Block 1 SD.
Mean Block 2 IBI is correlated with mean Block 2 VT, mean Block 2 CV and Mean Block 2 SD.

Table 14.
T-tests of Group Differences Associated with Different Cardiac Measures: Vagal Tone (VT), Interbeat Interval (IBI), Coefficient of Variation (CV), and Standard Deviation of IBI (SD).

	Cardiac Measure			
	VT t	*IBI* t	*CV* t	*SD* t
Task				
Baseline	5.15***	.74	3.88**	3.85**
Flanker	5.47***	.89	3.94**	3.78**
Source, Block1	5.75***	1.00	3.59**	3.66**
Source, Block 2	6.56***	.66	4.77***	4.35***

Note. ** $p < .01$; *** $p < .001$. Df = 26 for all t-tests.

Figure 1. Affective and cognitive divisions of anterior cingulate cortex. (Bush, Luu & Posner, 2000).

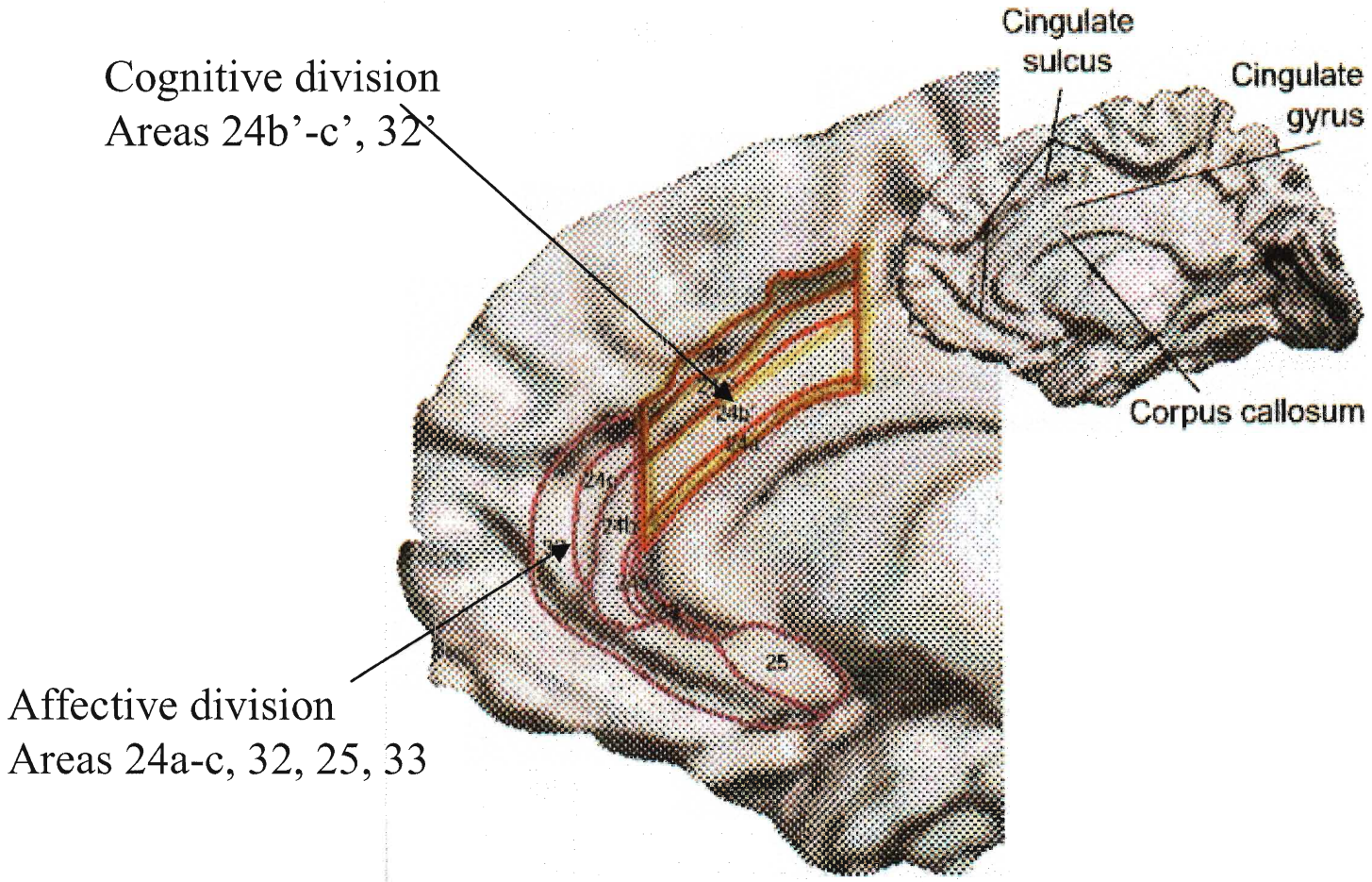


Figure 2. 36 Scalp electrode sites from which EEG was recorded.

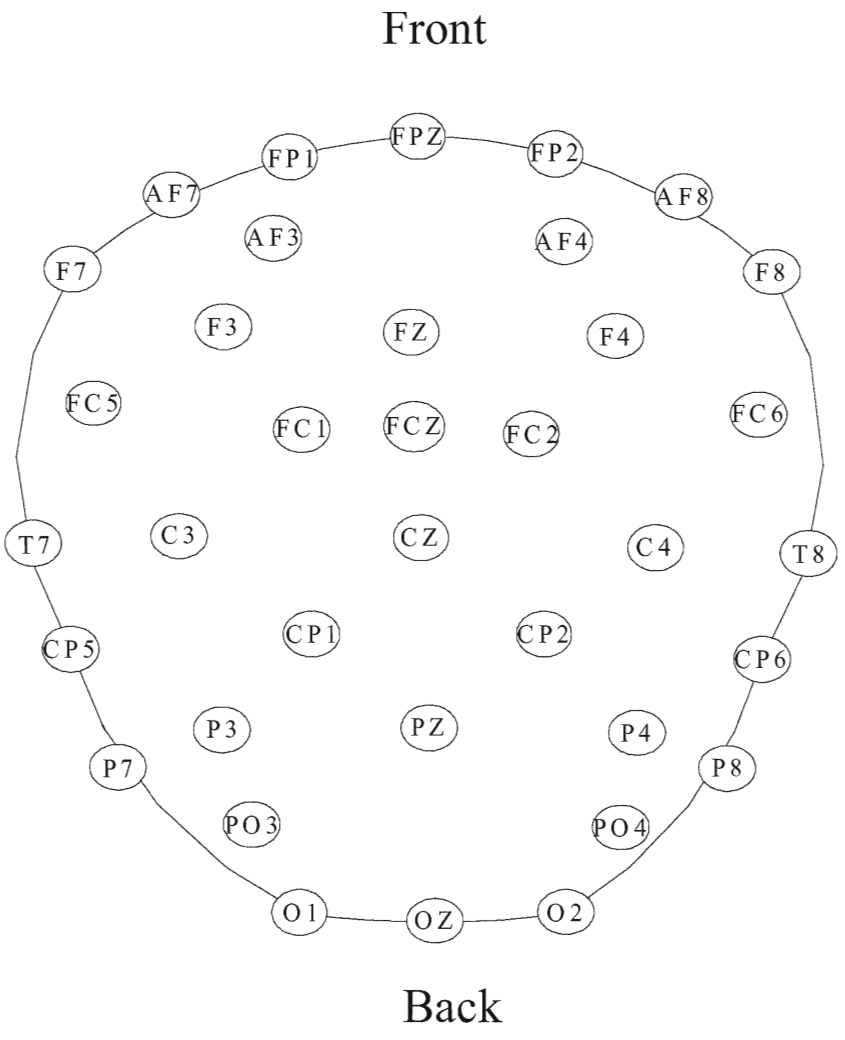


Figure 3. Mean errors for younger and older adults in the flanker task. N = 15 younger; N = 14 older.

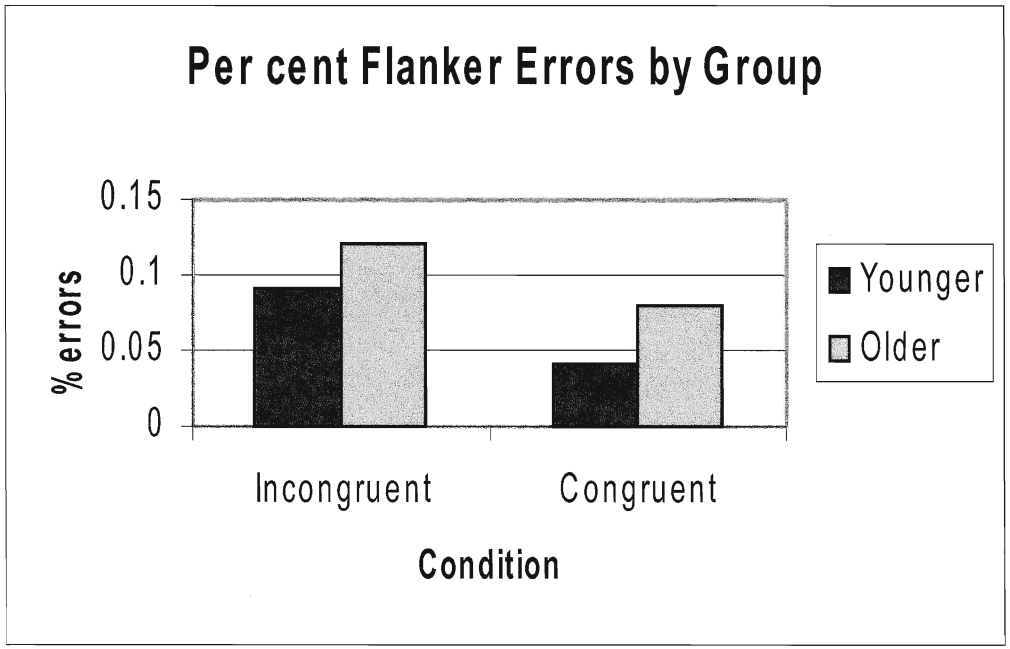


Figure 4. Mean response times for younger and older adults for incongruent and congruent trials in the flanker task. N = 15 younger; N = 14 older.

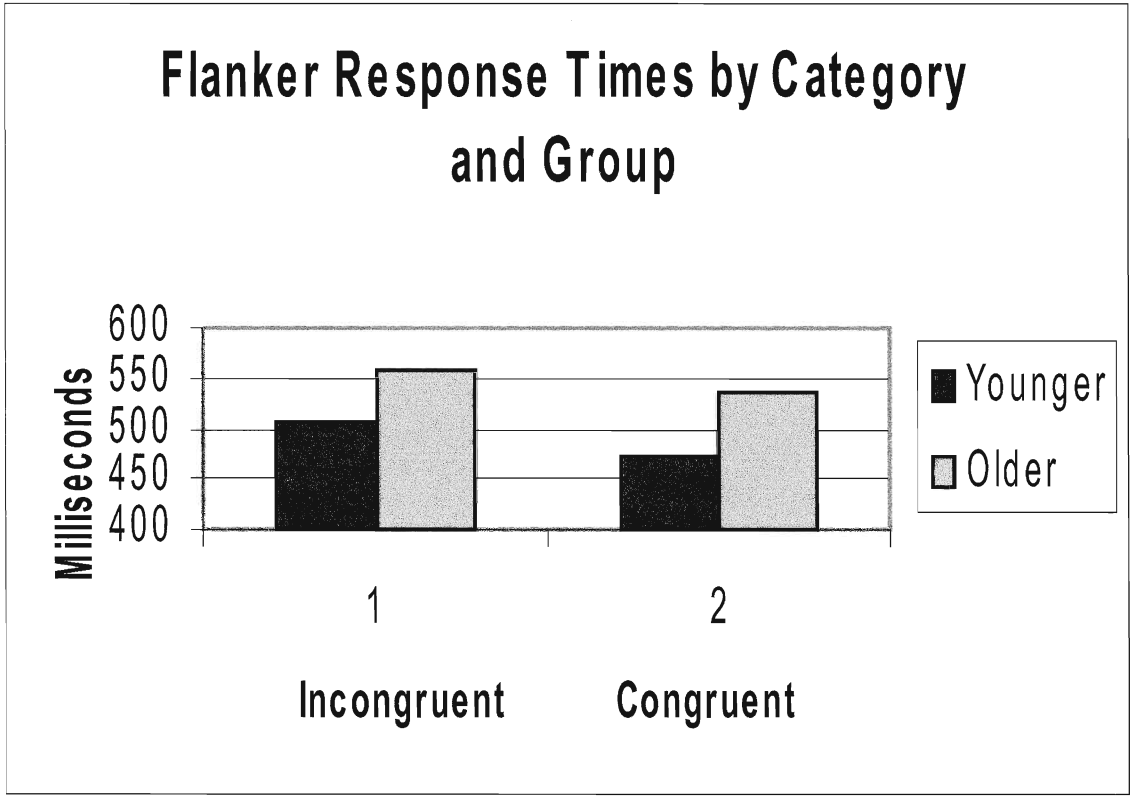


Figure 5. Grand average ERPs (unfiltered) from correct and incorrect responses in the flanker task for younger adults. N = 16.

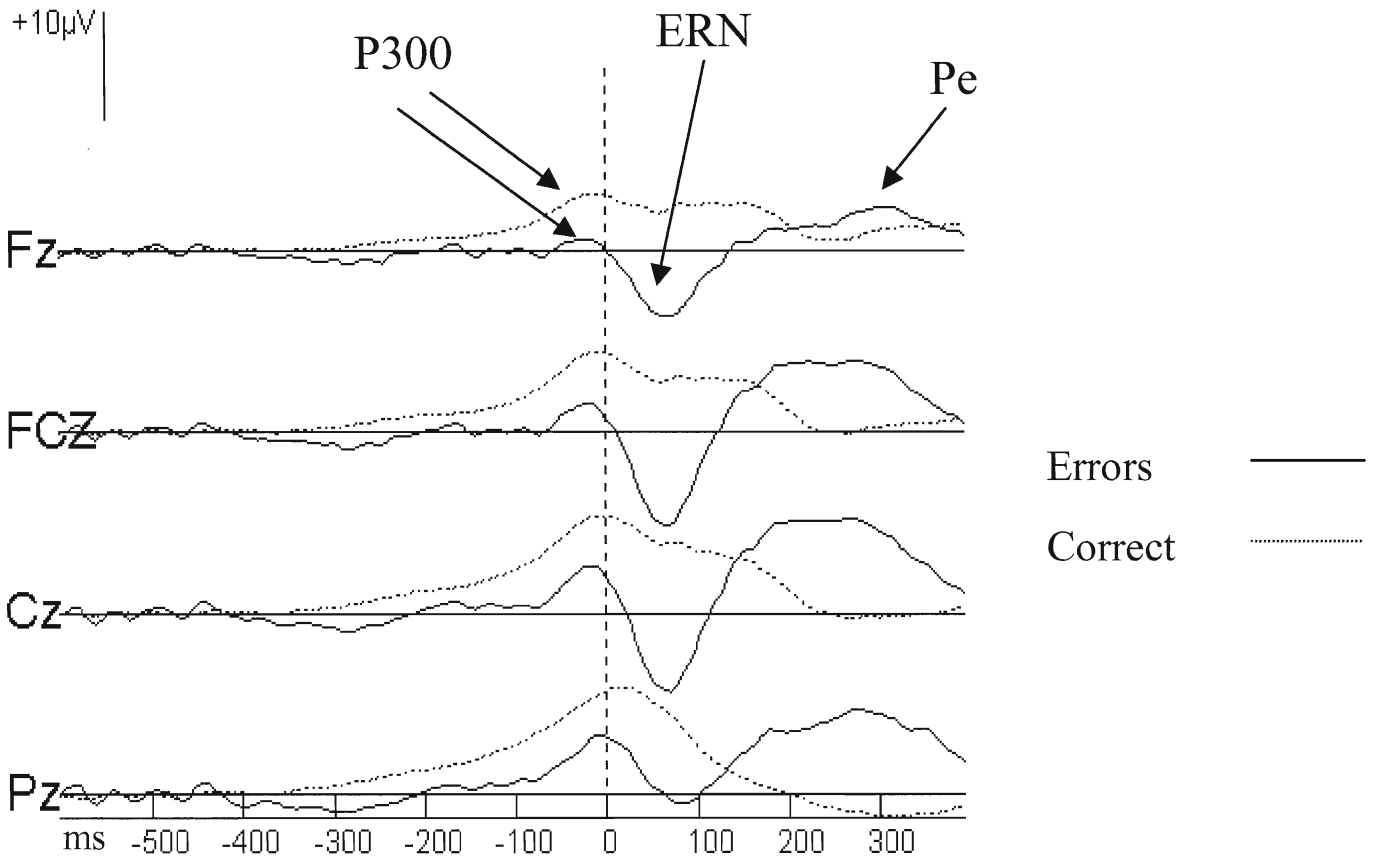


Figure 6. Grand average ERPs (unfiltered) from correct and incorrect responses in the flanker task for older adults. N = 16.

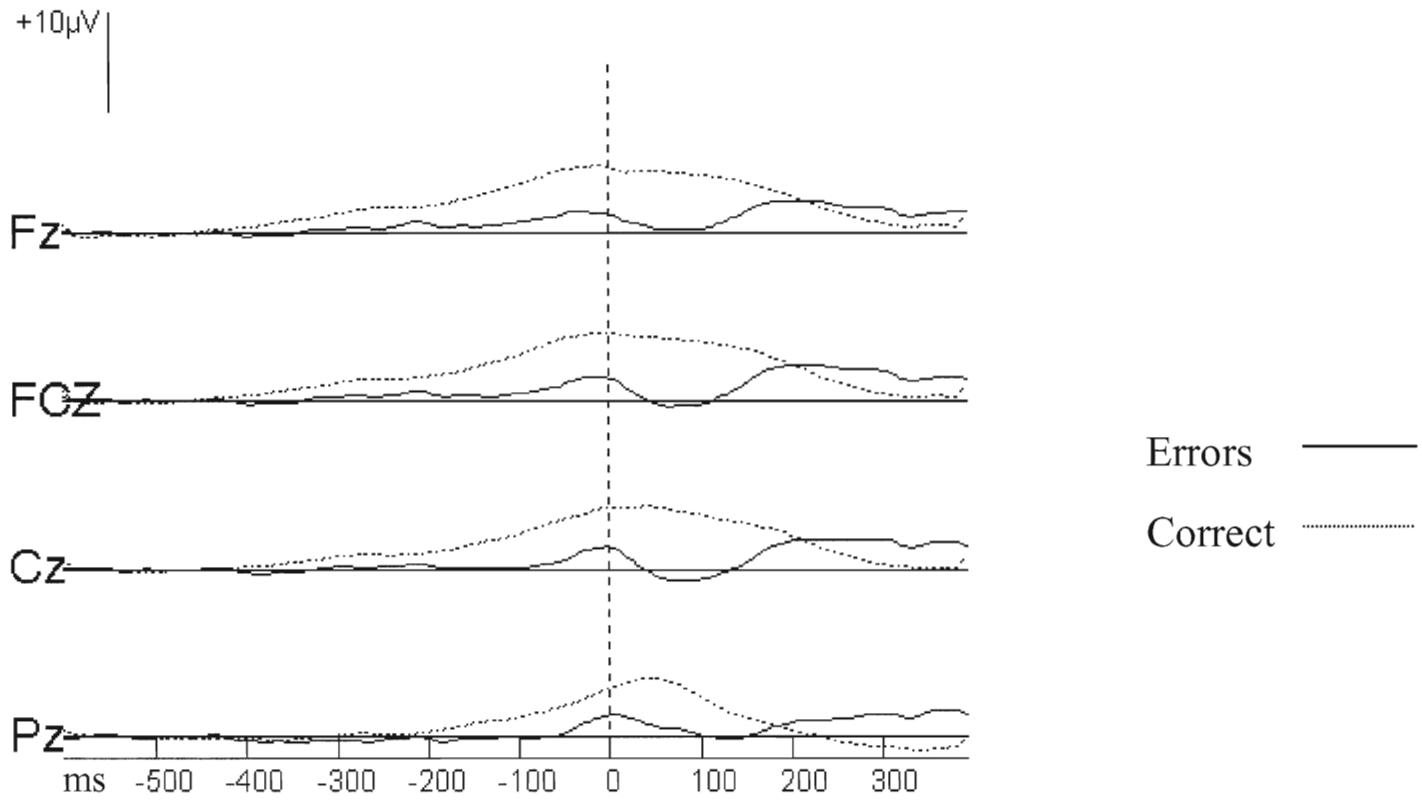


Figure 7. Grand average ERPs (unfiltered) from correct trials in the flanker task for younger and older adults.

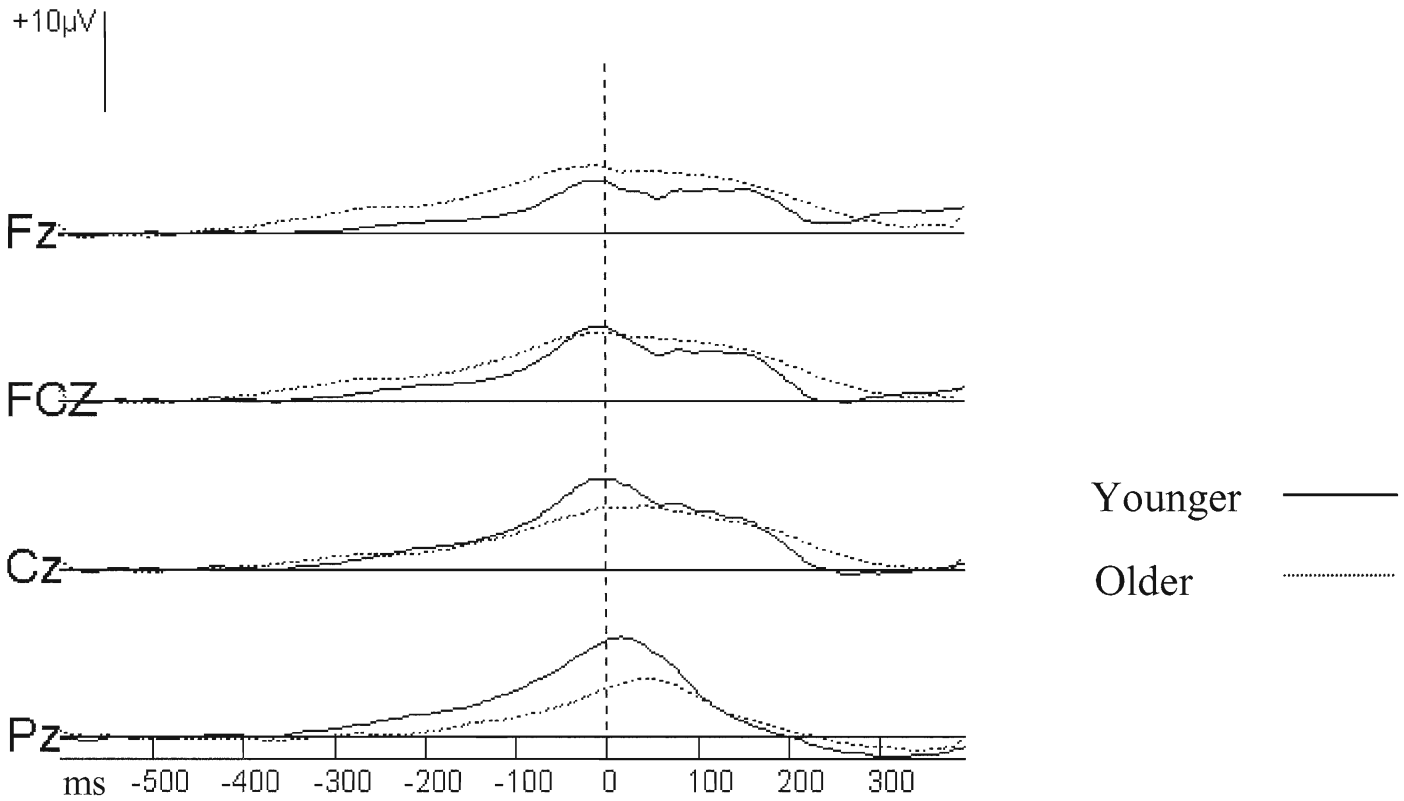


Figure 8. Grand average ERPs (unfiltered) from error trials in the flanker task for younger and older adults.

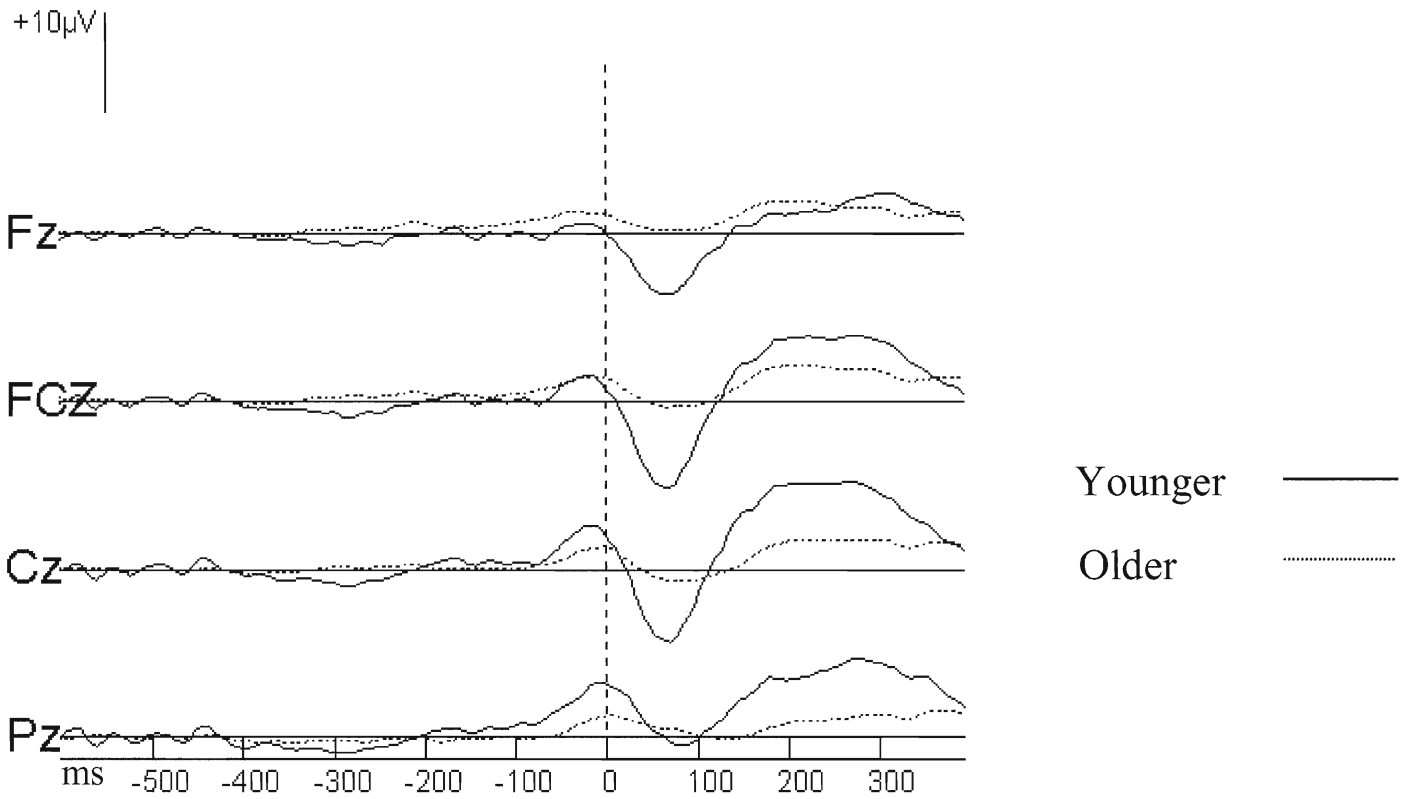


Figure 9. Grand average ERP waveforms (unfiltered) for error trials in the flanker task after subtracting waveforms for correct trials. (Difference wave for younger and older adults).

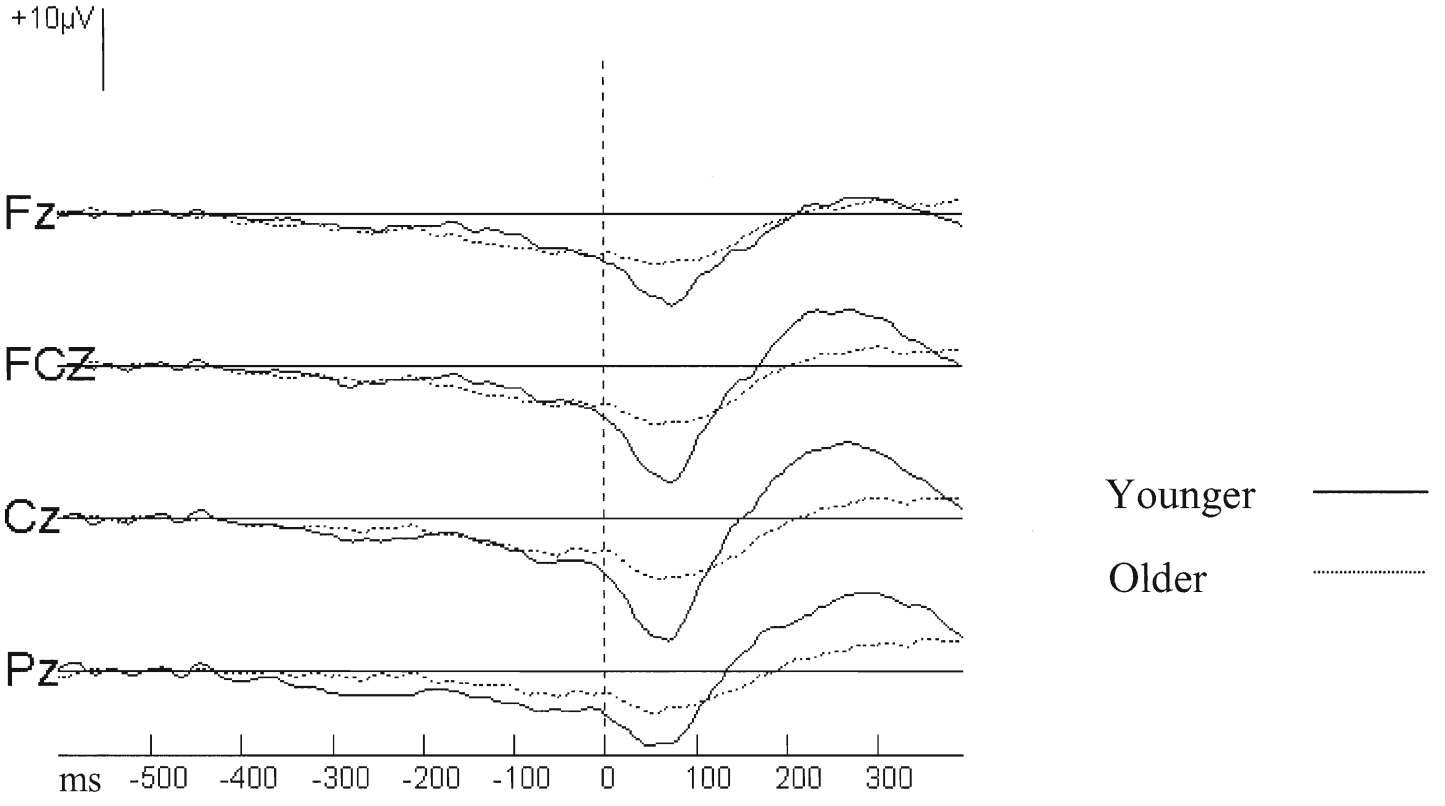


Figure 10. Mean ERP amplitudes elicited by error responses in the flanker task for younger and older adults.

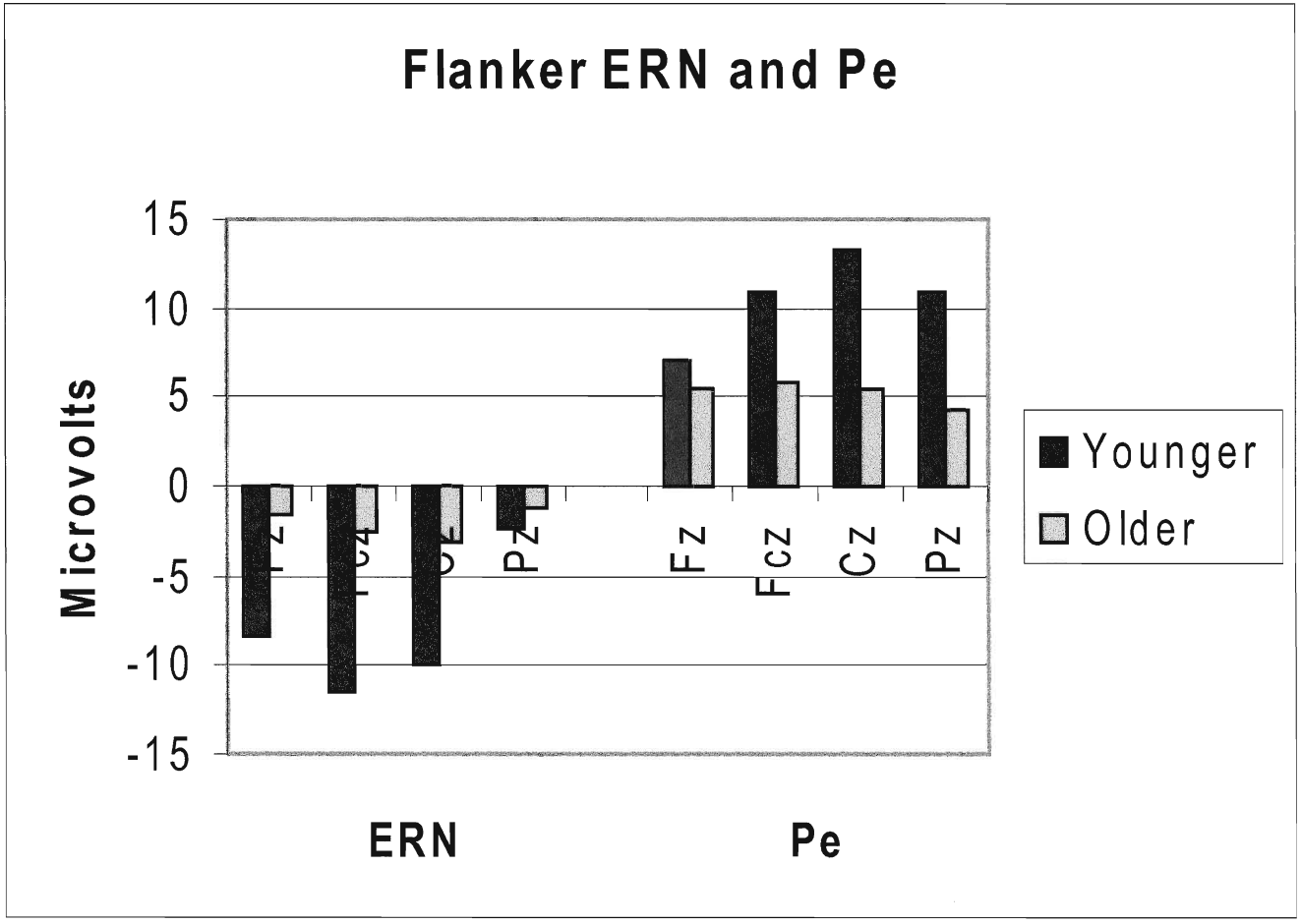


Figure 11. Mean proportion of words called "study" for the different word-types in the source memory task.

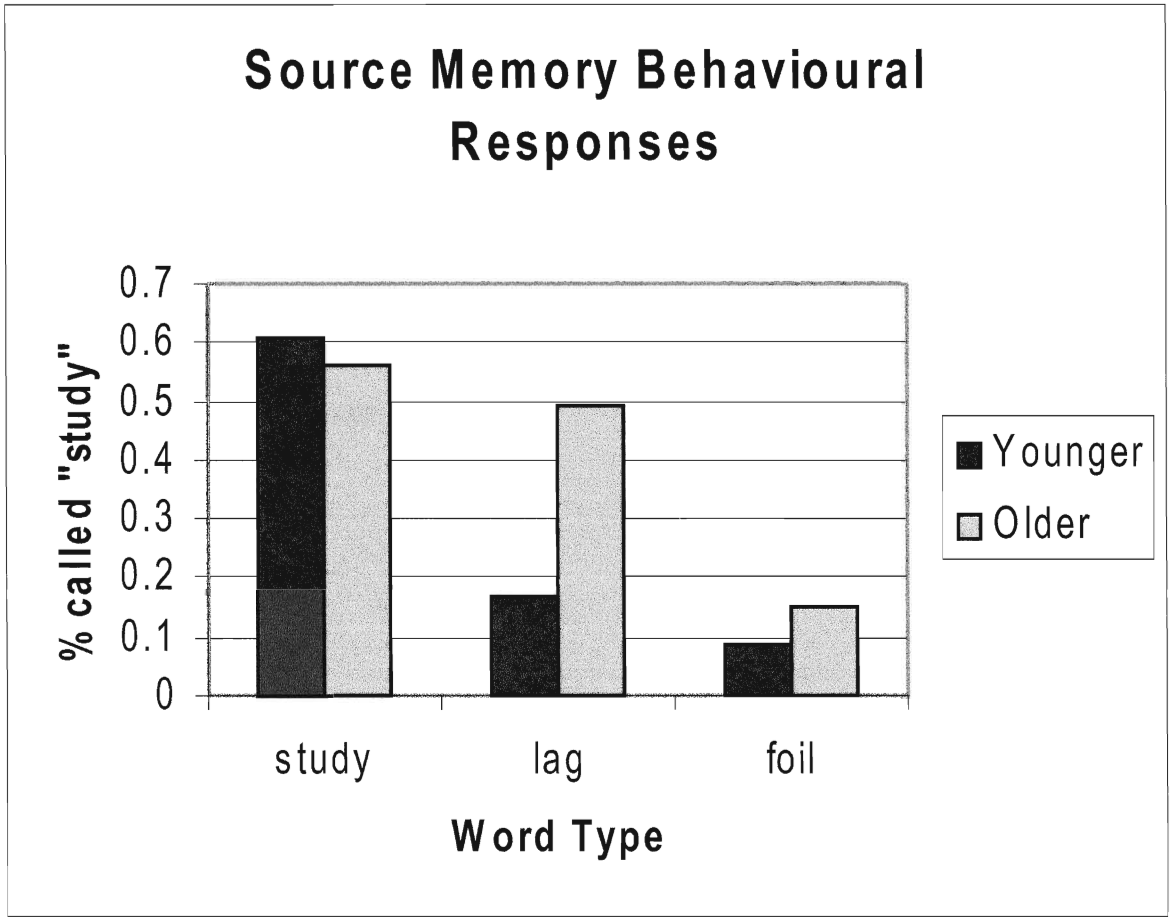


Figure 12. Correlations between the number of WCST categories completed and the number of errors made on lag words in the source memory task.

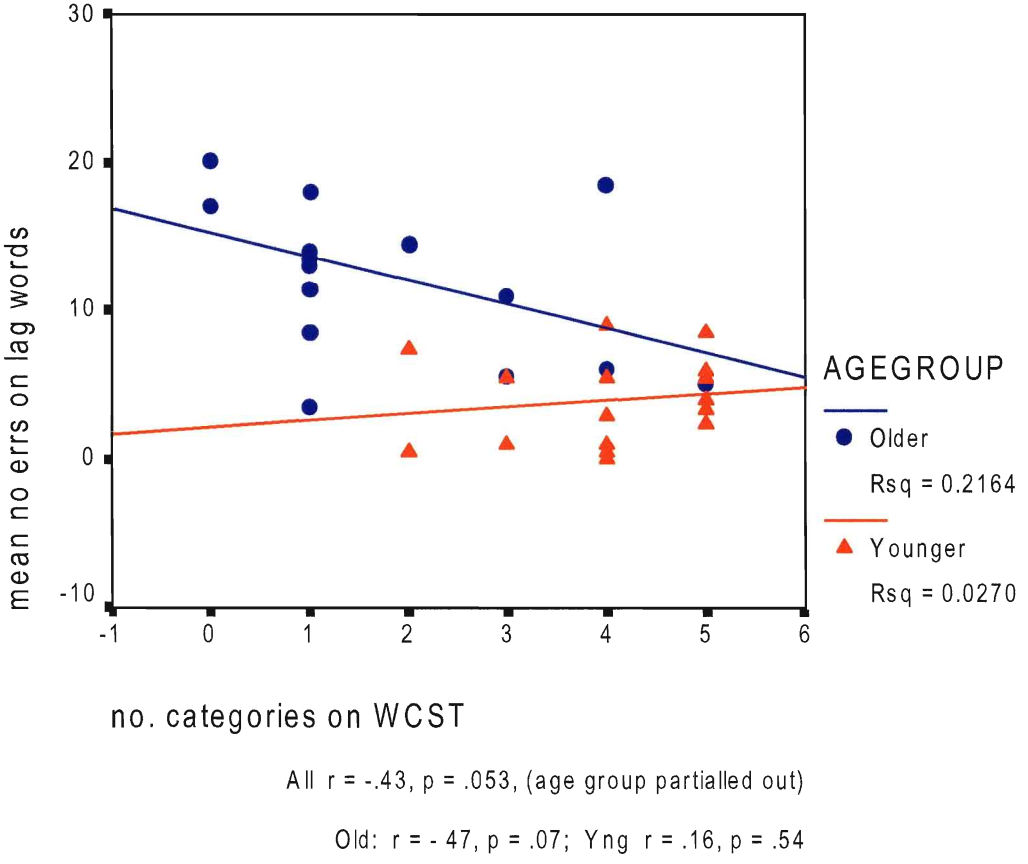


Figure 13. Grand average stimulus-locked ERPs (21 point filter) generated to study, lag and foil words in the source memory task, by group. N = 32.

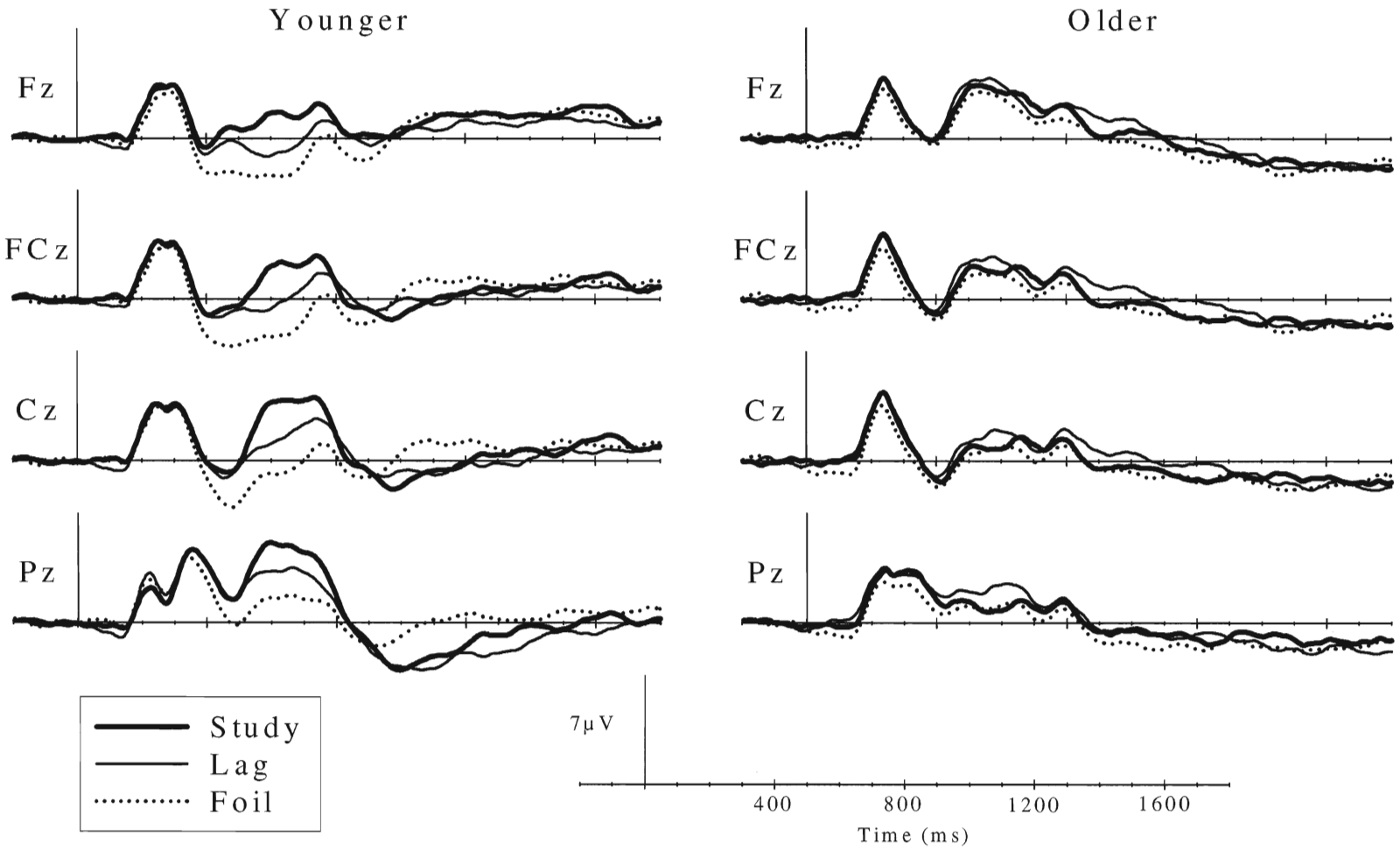


Figure 14. Grand average response-locked ERPs (7-pt filter) for correct and incorrect responses to lag words from the source memory task for younger adults. N = 9.

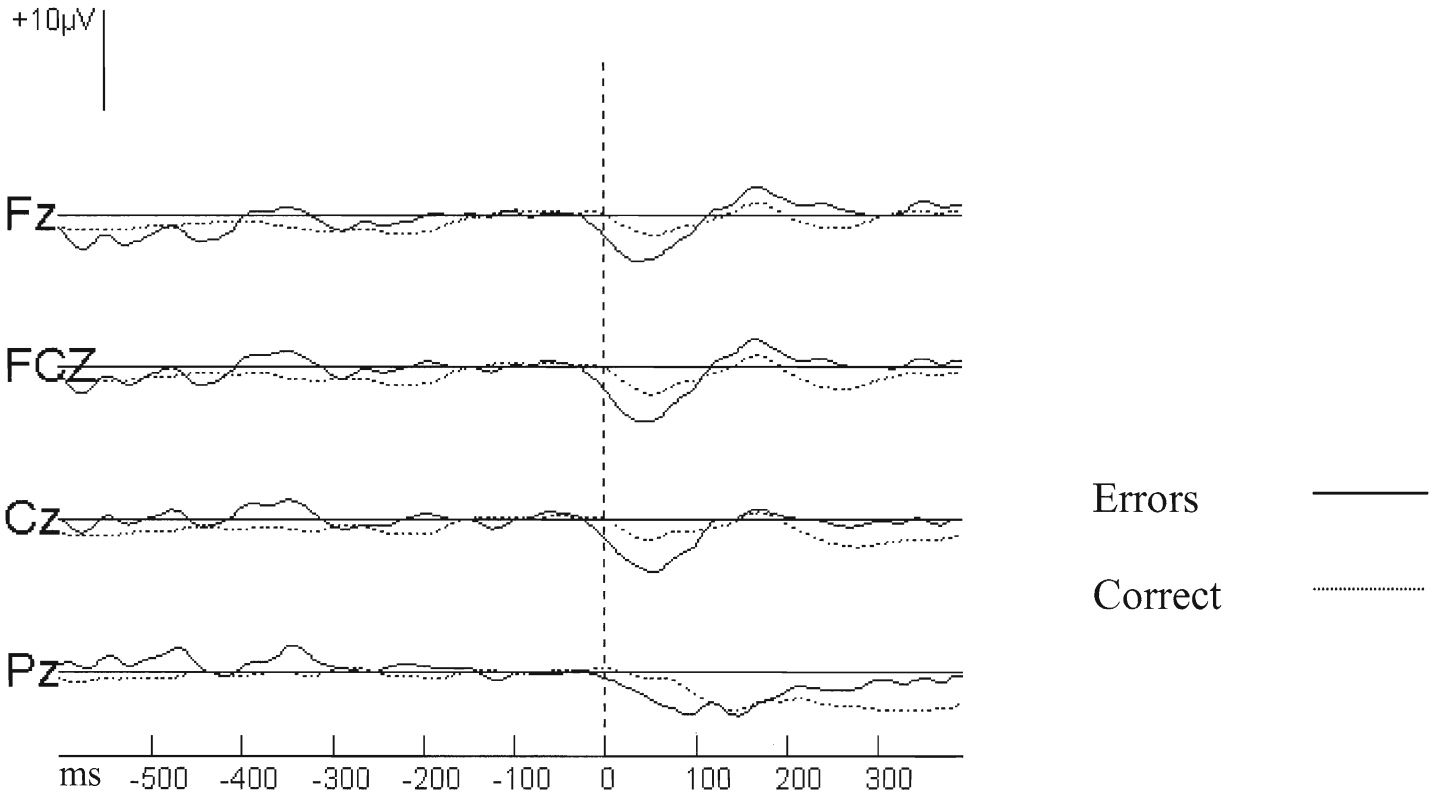


Figure 15. Grand average response-locked ERPs (unfiltered) for correct and incorrect responses to lag words from the source memory task for older adults. N = 16.

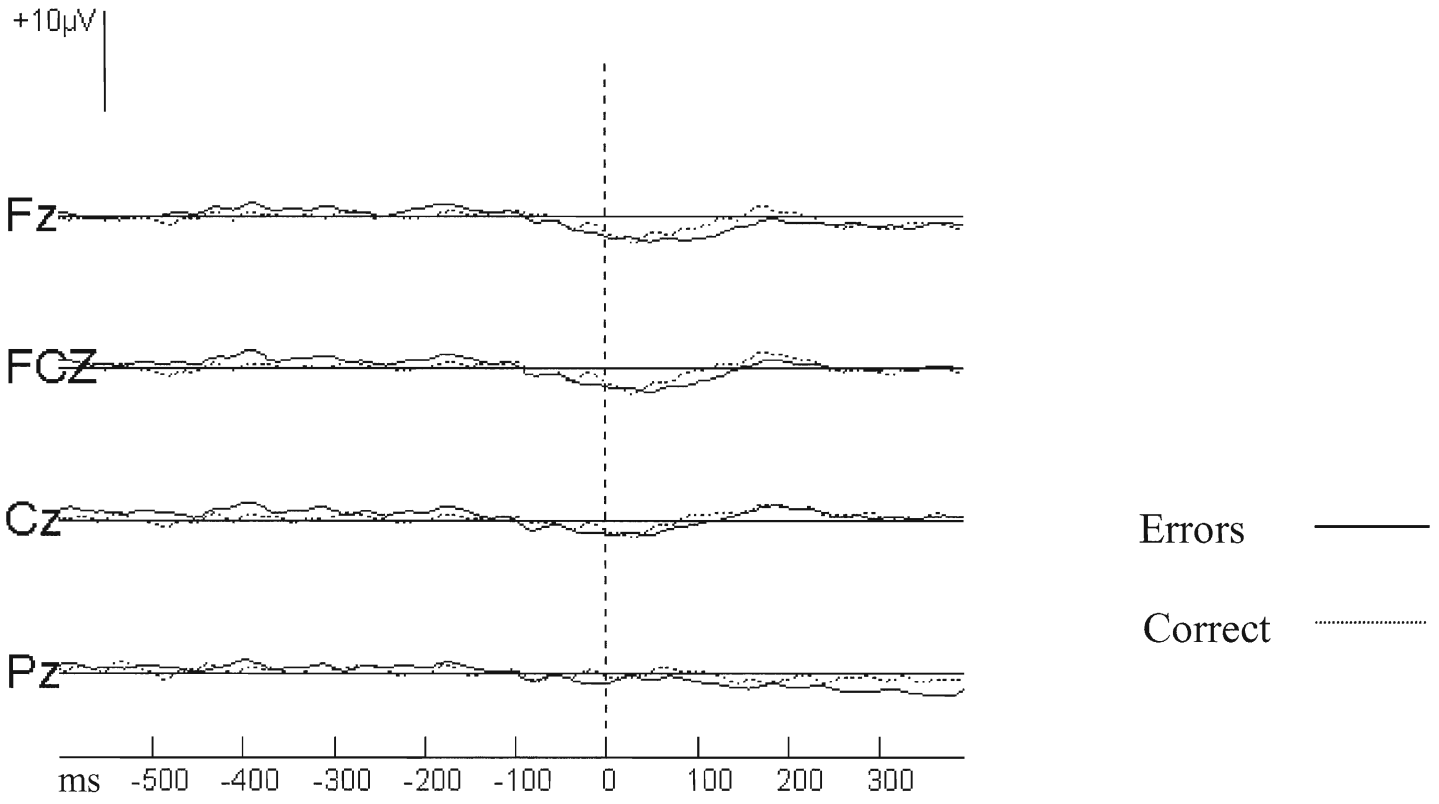


Figure 16. Grand average response-locked ERPs (7-pt filter) for error responses to lag words from the source memory task for younger and older adults.

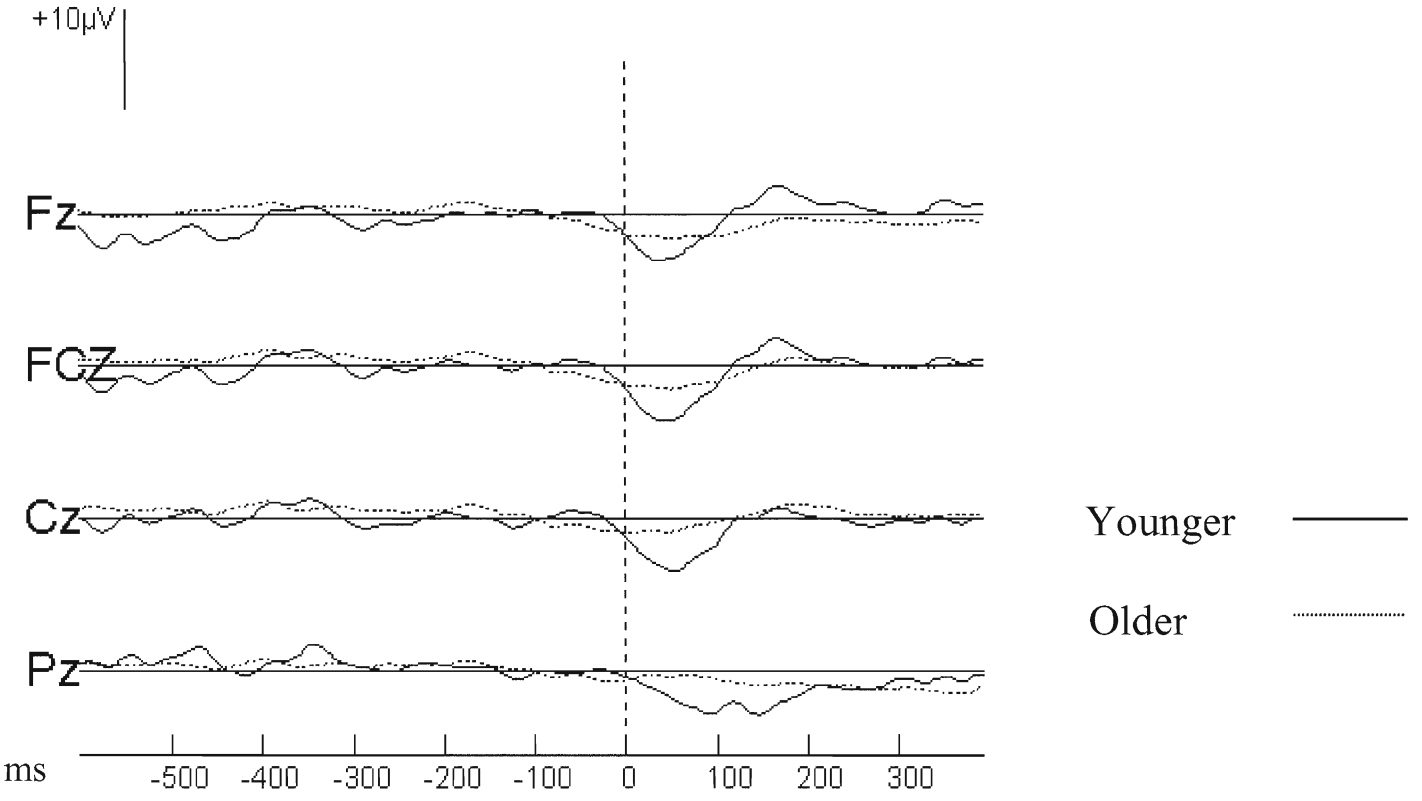


Figure 17. Mean ERN amplitudes from the flanker task and source memory task by group and site.

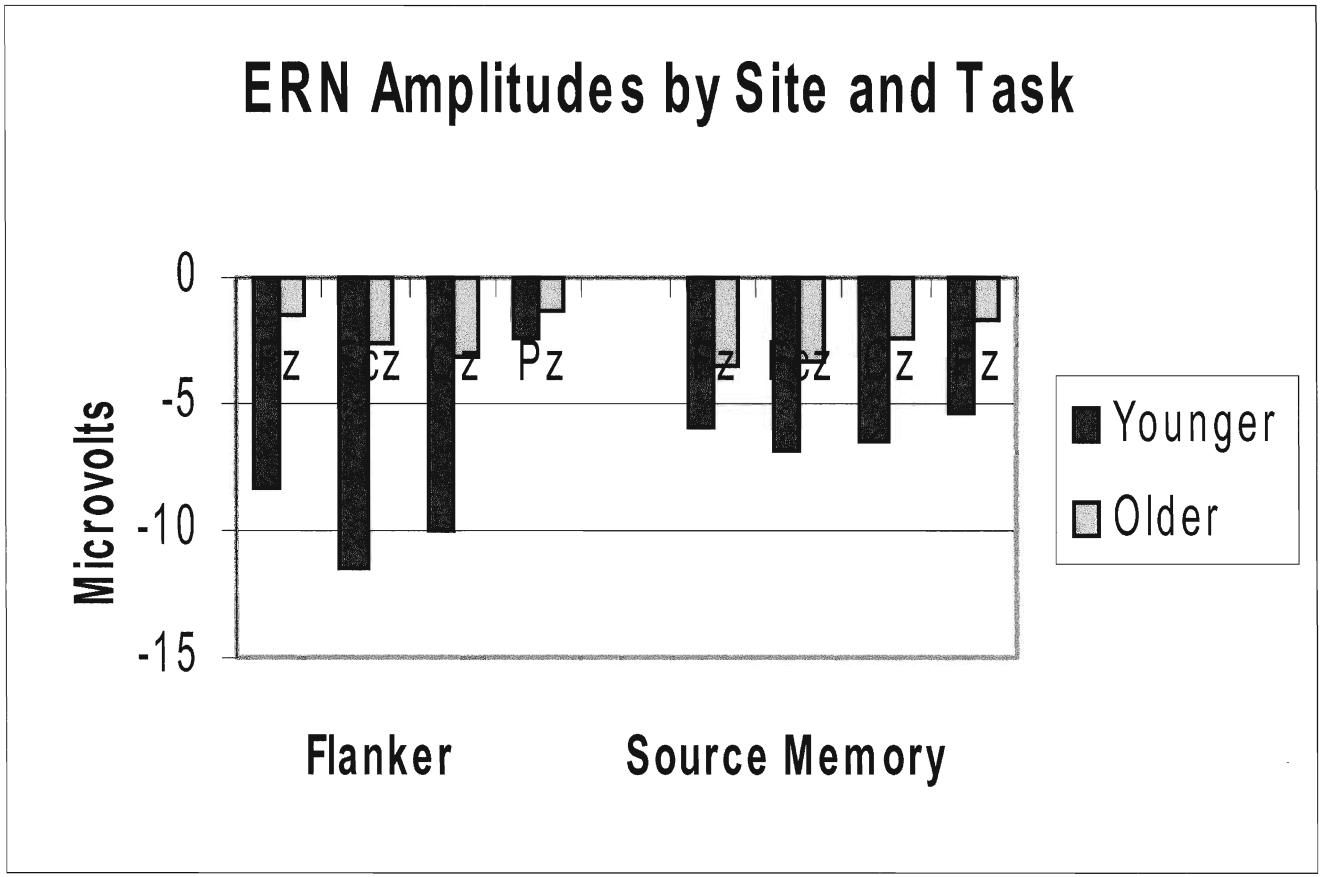


Figure 18. Mean Pe amplitudes from the flanker task and source memory task by group and site.

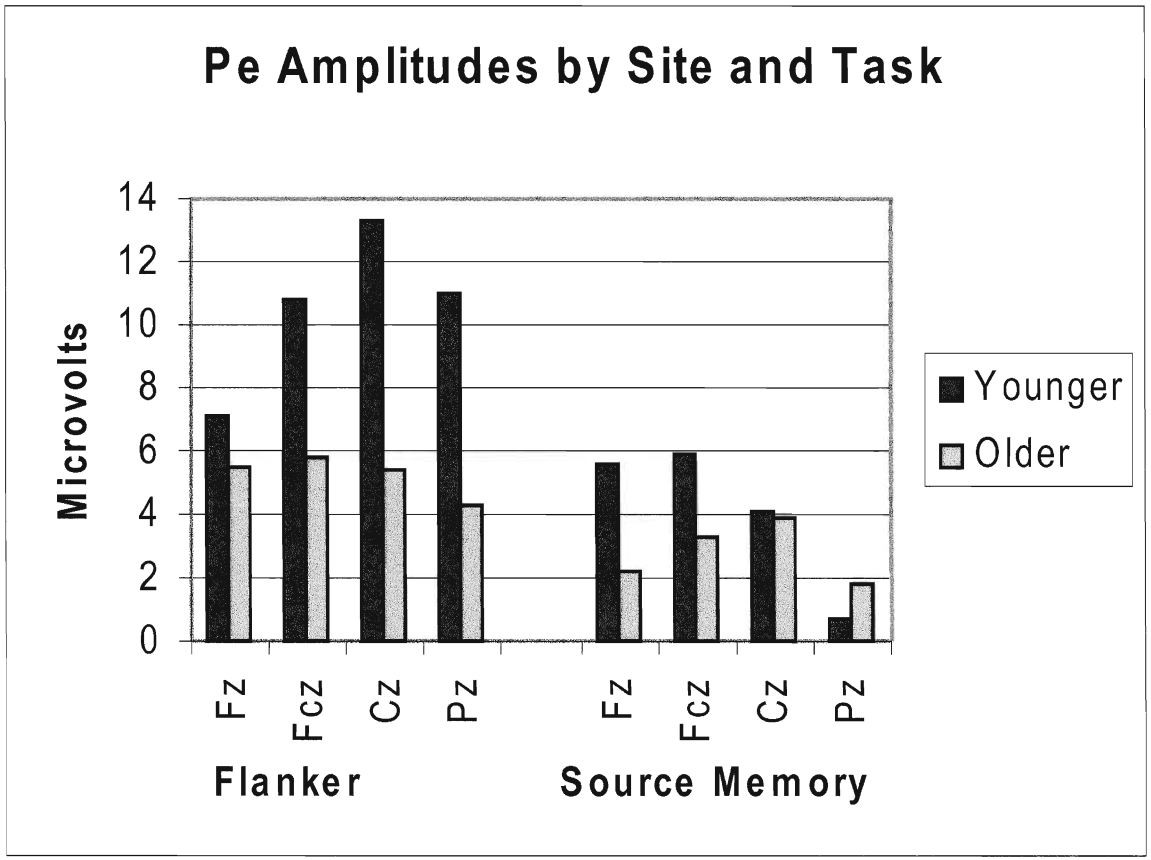
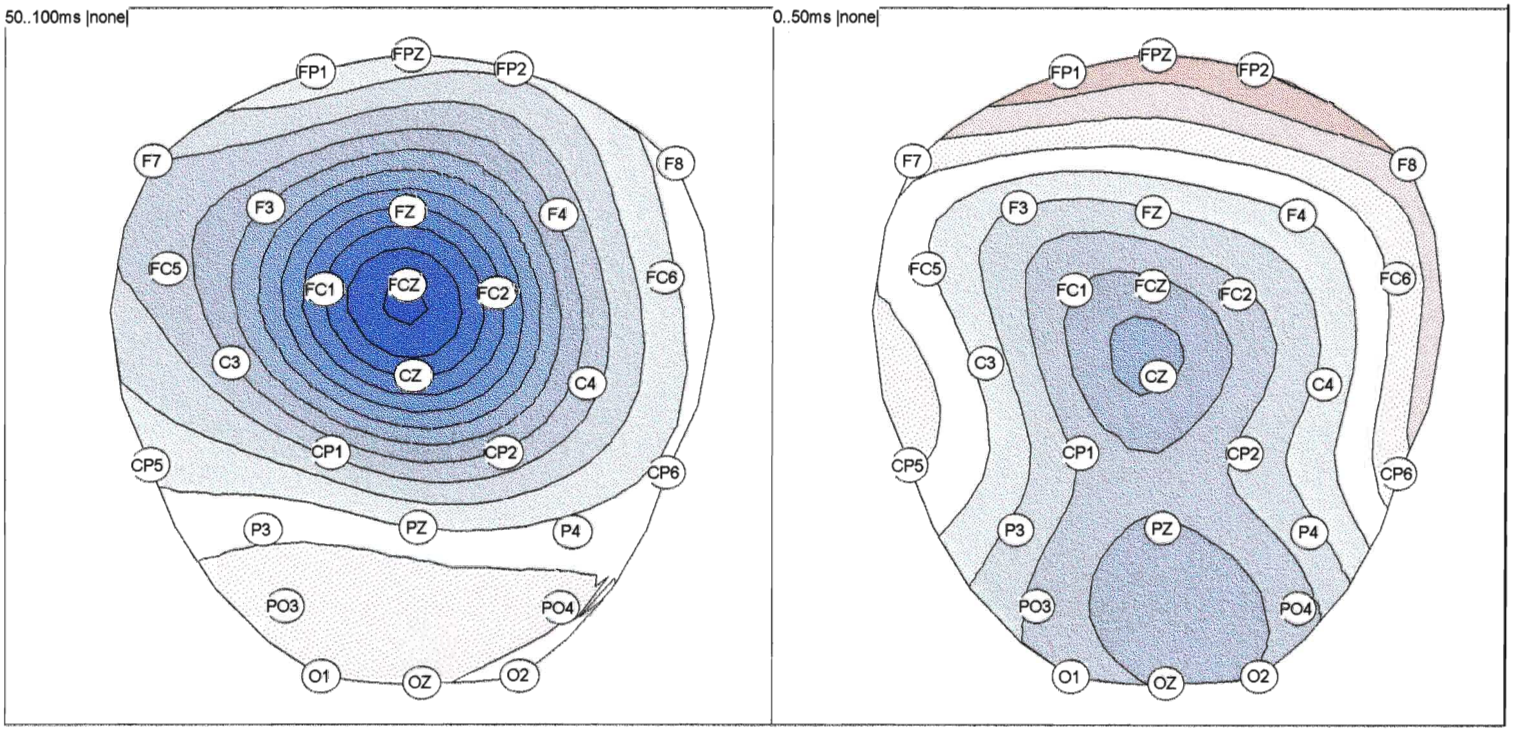


Figure 19. Topographical comparisons of ERN amplitude in the flanker task (left) and the source memory task (right) in young adults. Blue is negative; red is positive. Range = -7.0 to 9.0 microvolts.

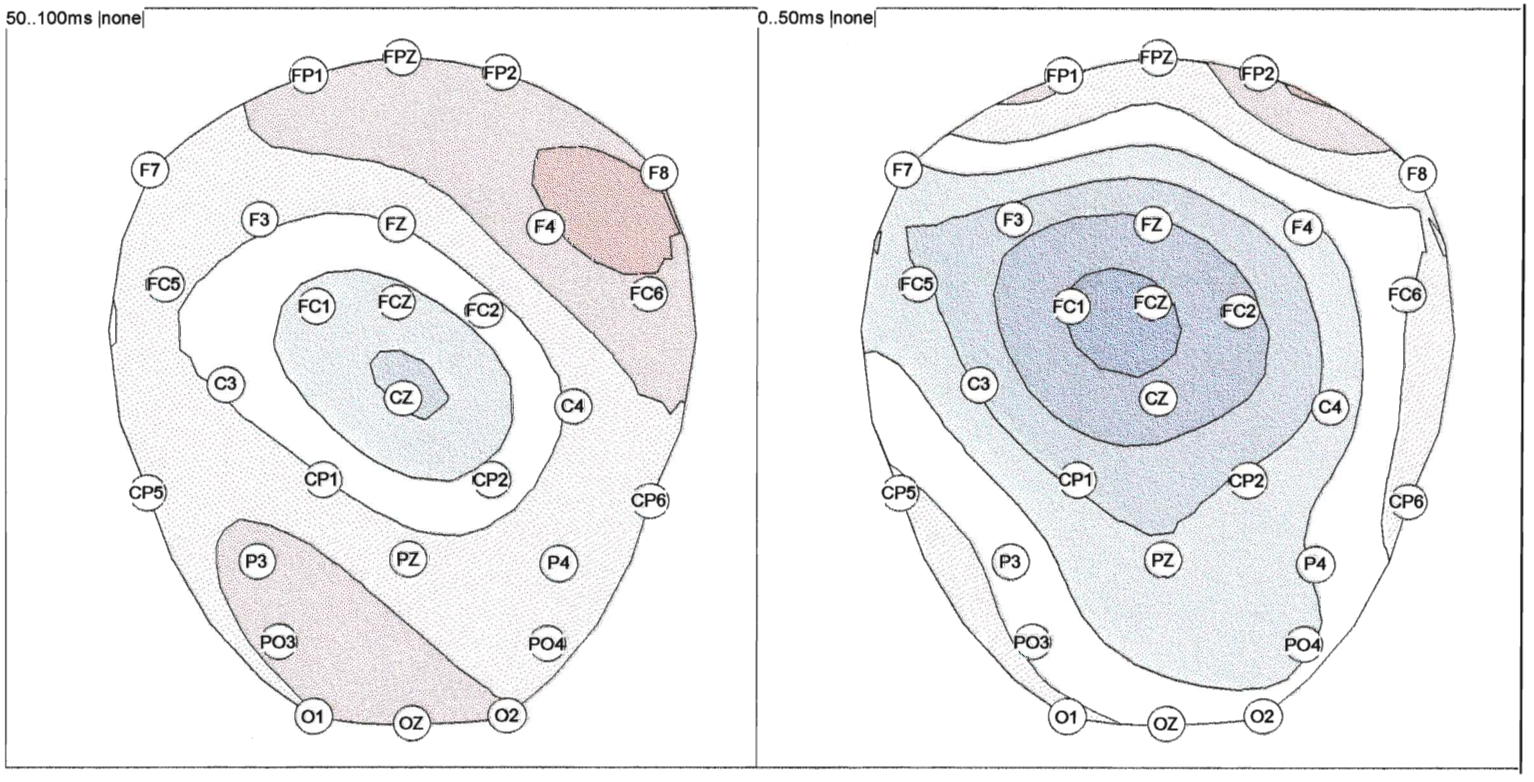
Front



Back

Figure 20.Topographical comparisons of ERN amplitude in the flanker task (left) and the source memory task (right) in older adults. Blue is negative; red is positive. Range = -7.0 to 9.0 microvolts.

Front

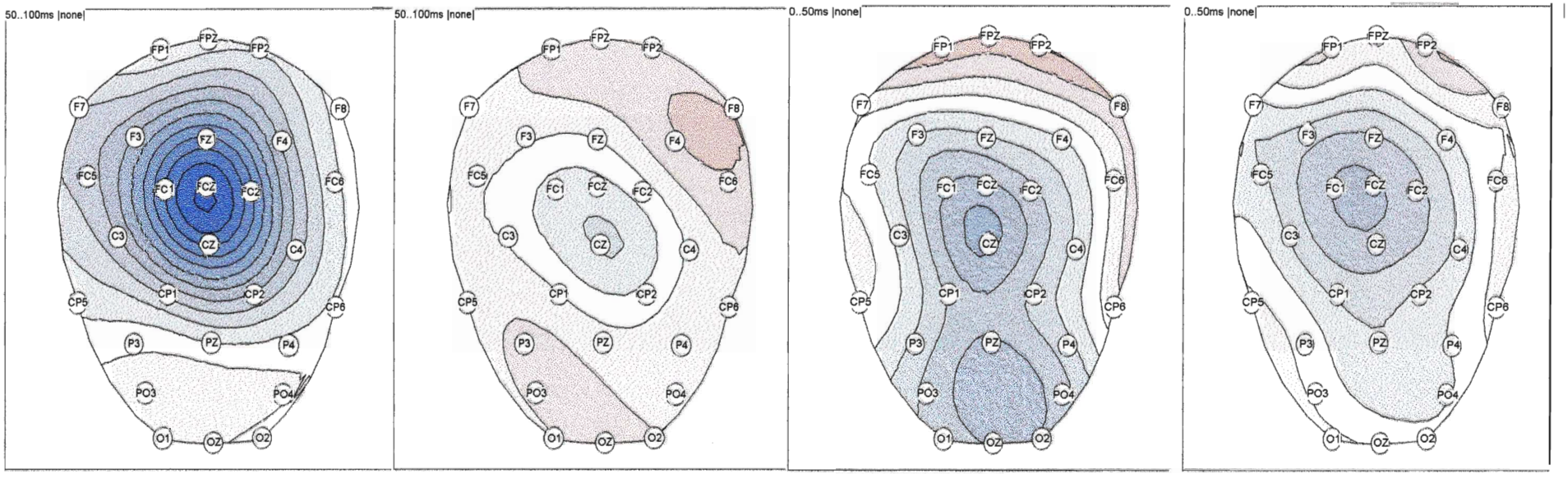


Back

Figure 21. Topographical comparisons of ERNs from both tasks and groups. Blue is negative; red is positive. Range = -7.0 to 9.0 microvolts.

Flanker

Source Memory



Young

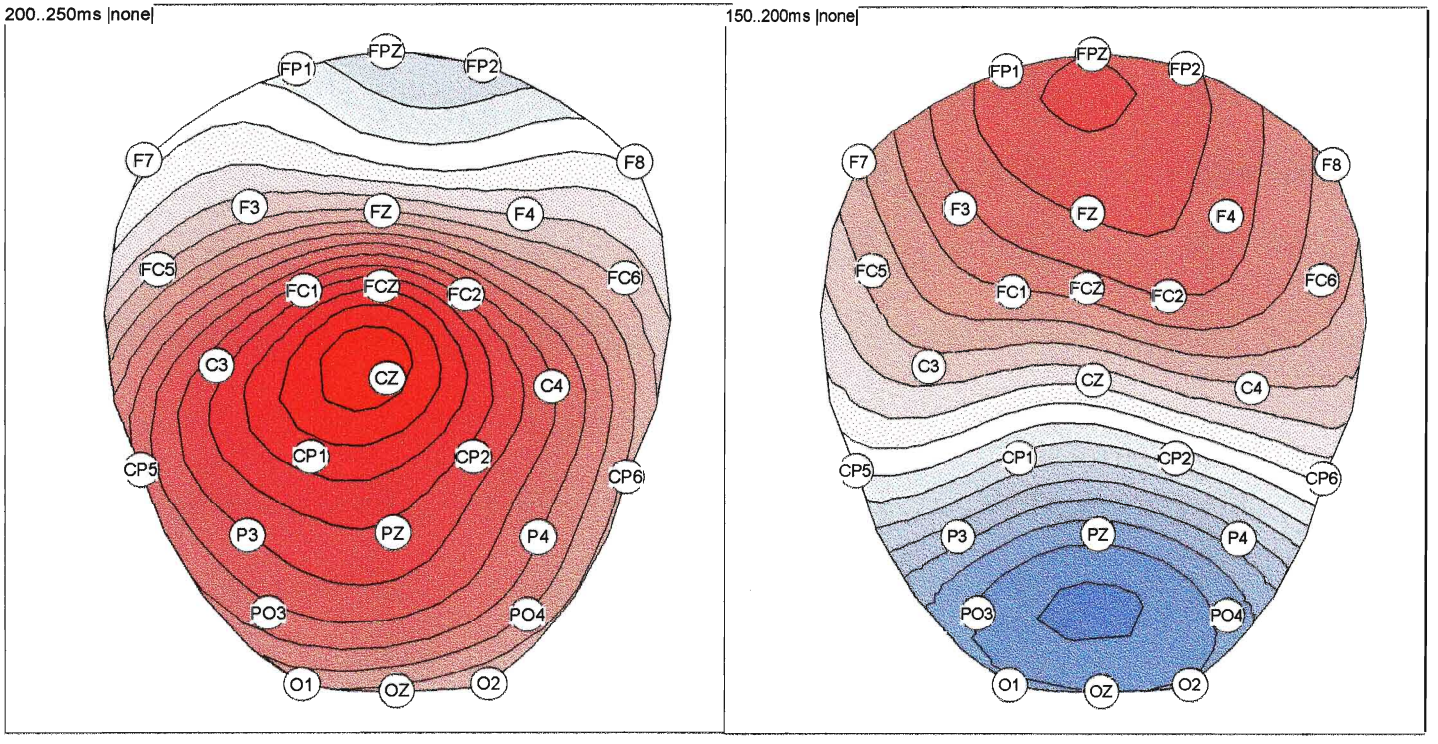
Older

Young

Older

Figure 22. Topographical comparisons of Pe amplitude in the flanker task (left) and the source memory task (right) in young adults. Blue is negative; red is positive. Range = -7.0 to 9.0 microvolts.

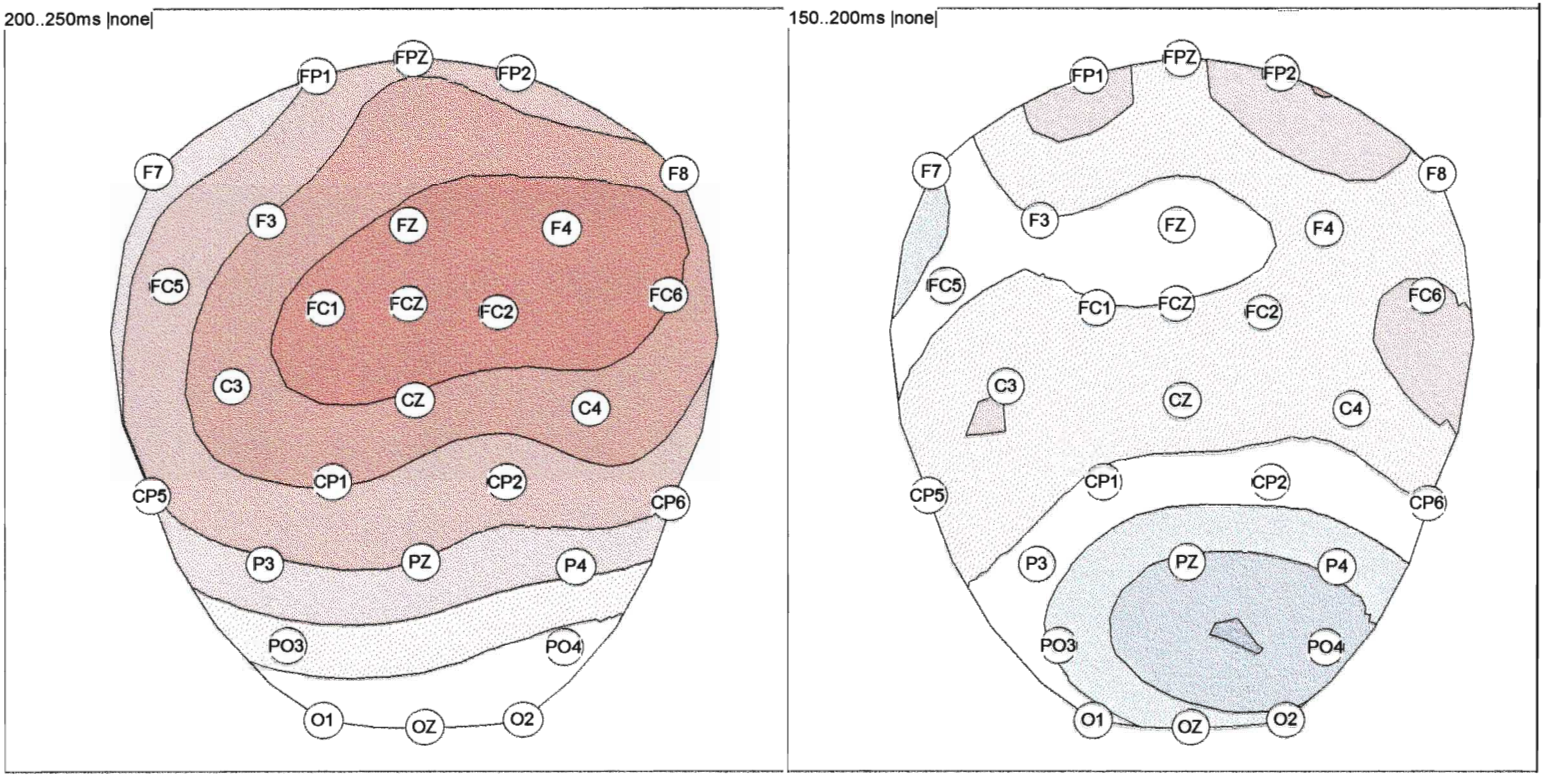
Front



Back

Figure 23. Topographical comparisons of Pe amplitude in the flanker task (left) and the source memory task (right) in older adults. Blue is negative; red is positive. Range = -7.0 to 9.0 microvolts.

Front

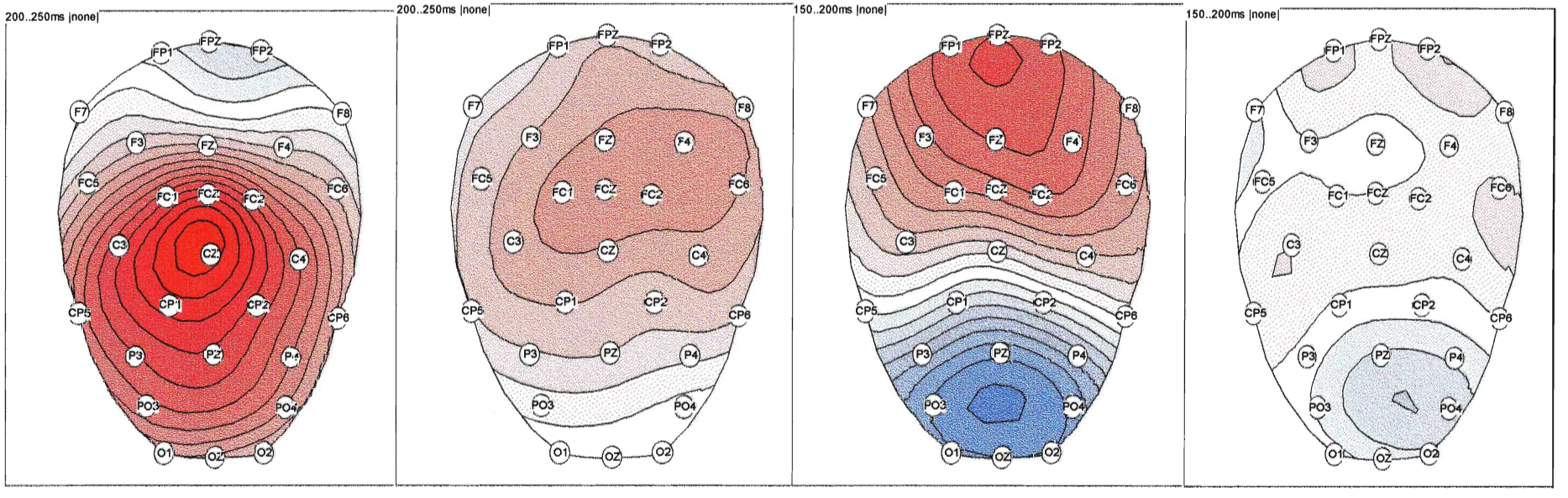


Back

Figure 24. Topographical comparisons of Pe's from both tasks and age groups. Blue is negative; red is positive. Range = -7.0 to 9.0 microvolts.

Flanker

Source Memory



Young

Older

Young

Older

Figure 25. Mean P3 amplitude from the flanker task and mean LP amplitude from the source memory task by group and site.

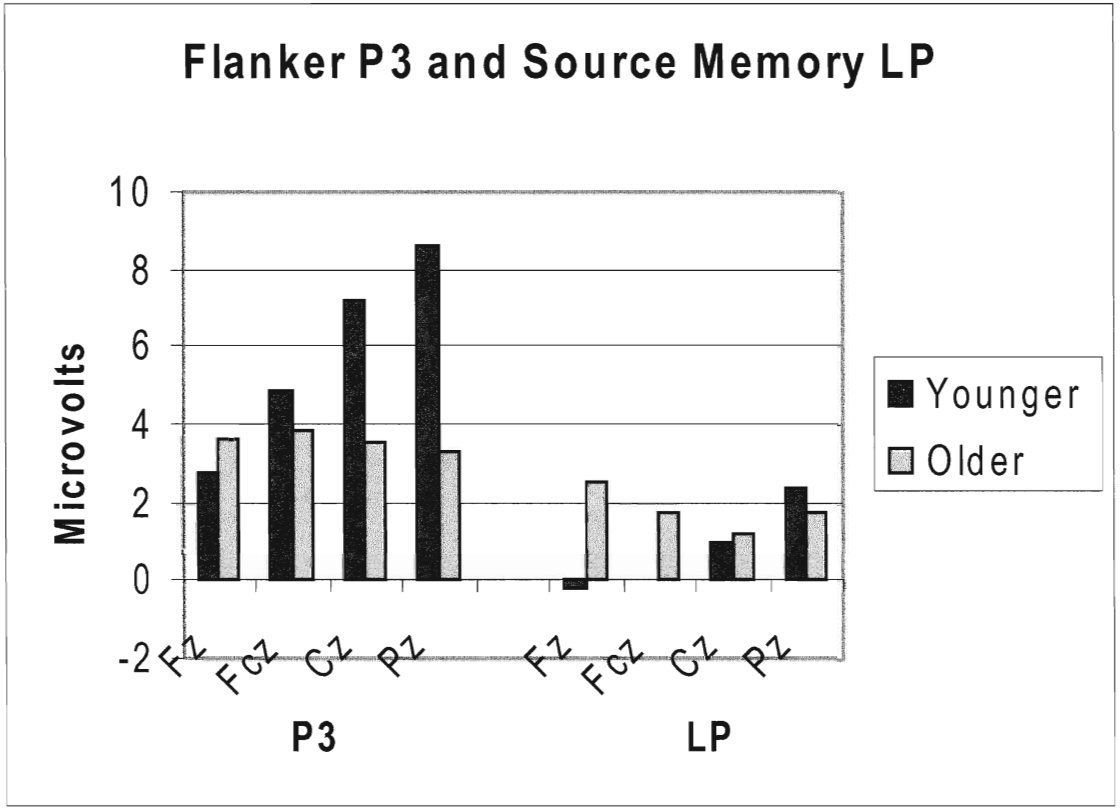


Figure 26. Topographical comparisons of P3 amplitude in the flanker task between younger adults (left) and older adults (right). Blue is negative; red is positive. Range = -7.0 to 9.0 microvolts.

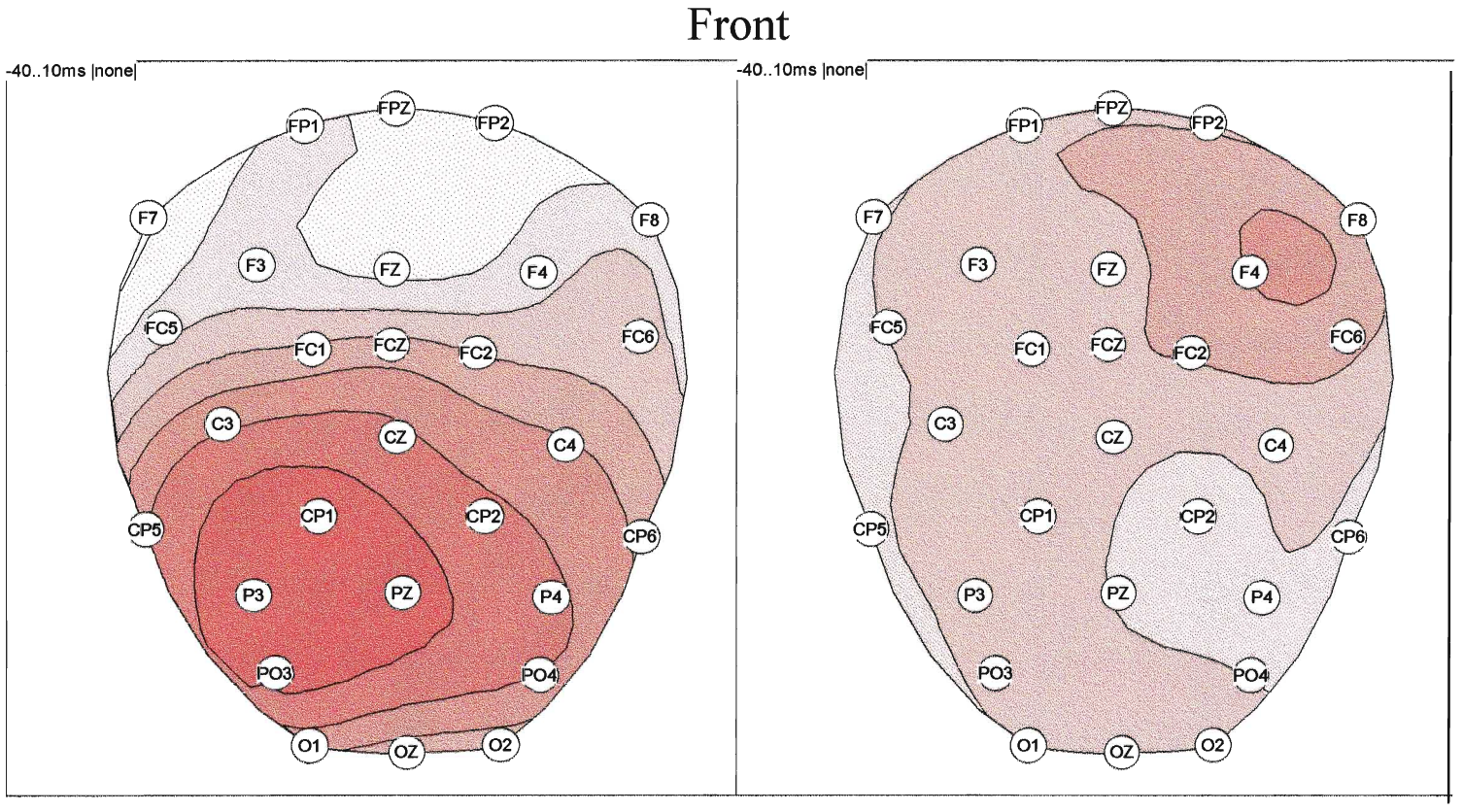


Figure 27. Mean vagal tone measures by condition and group. The VT values represent natural logarithms of the heart period variance that is associated with respiration.

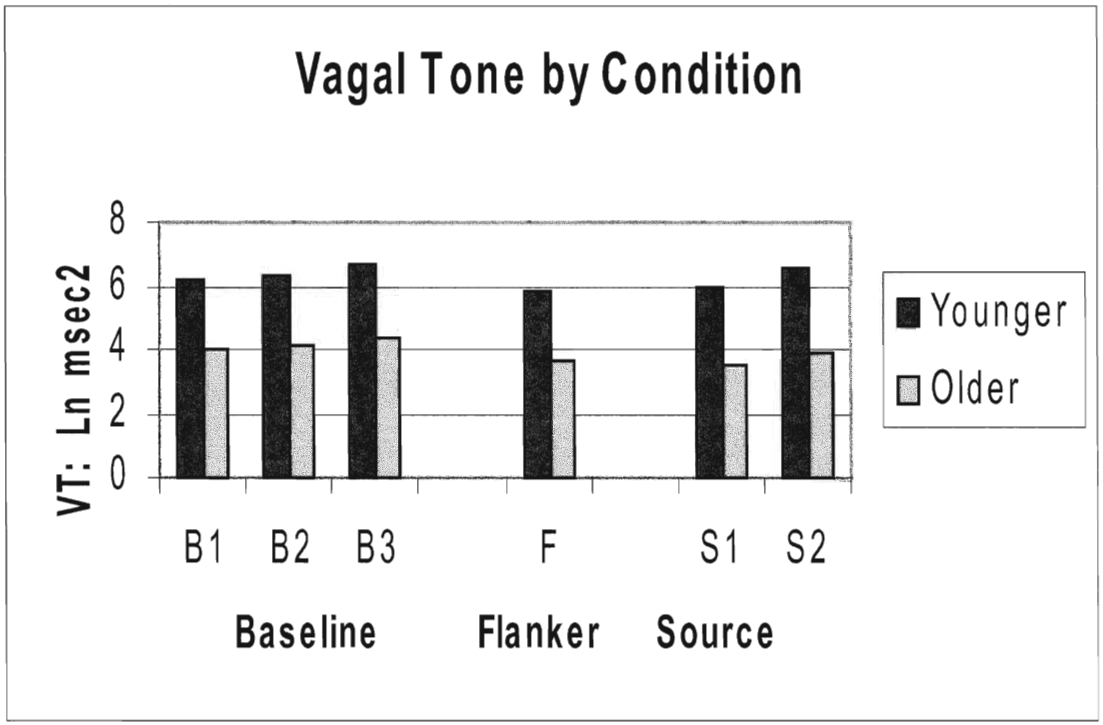


Figure 28. The interaction of the correlations between baseline vagal tone and errors from the source memory task, for each age group. This interaction marginally predicts ERN amplitude in the source memory task for the older group. (N = 29).

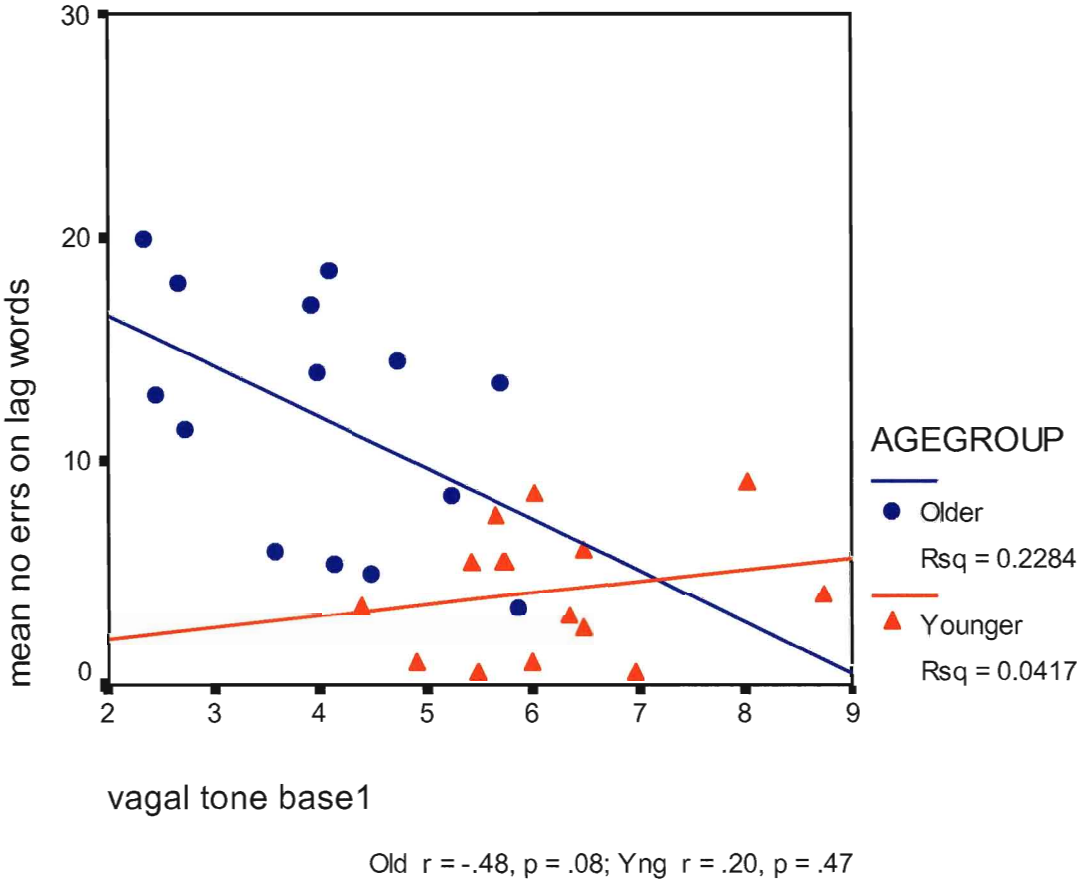


Figure 29. Prediction of mean Pe amplitude from the flanker task by residual vagal tone (Vtresid; N = 28).

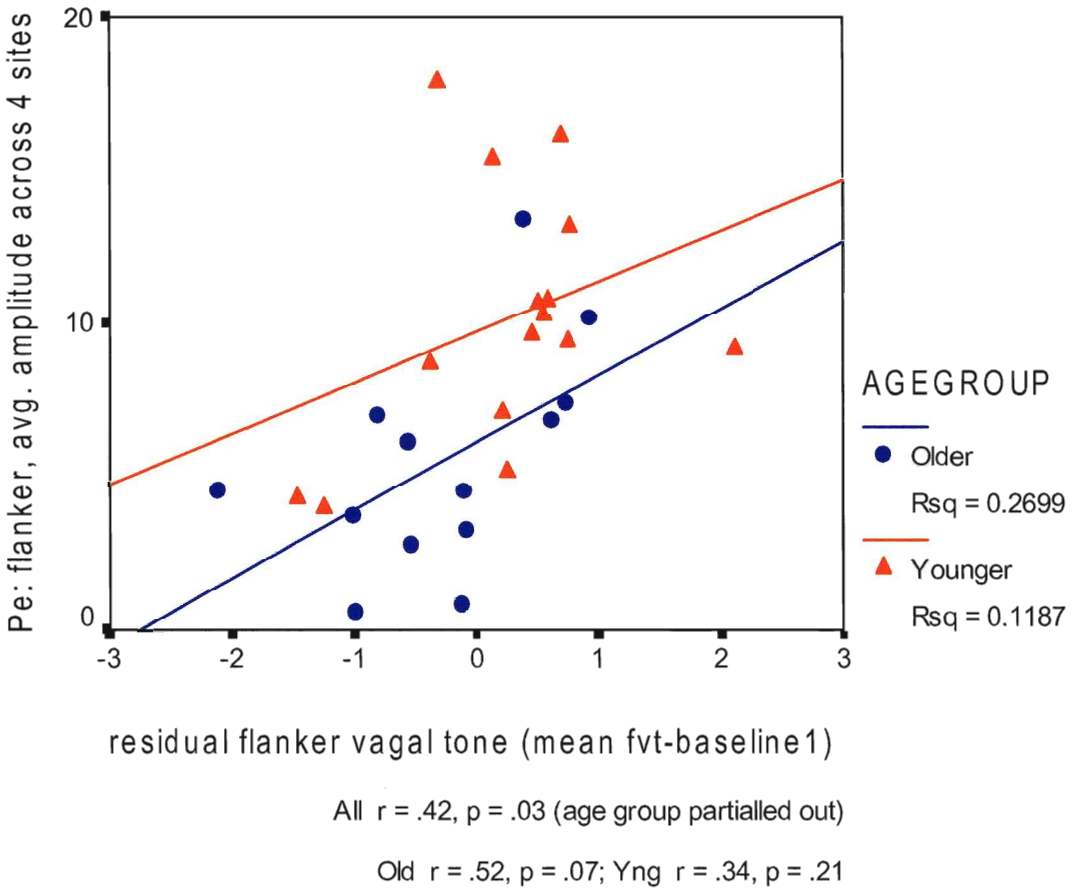


Figure 30. Prediction of mean ERN amplitude from the source memory task by baseline vagal tone. (N = 23).

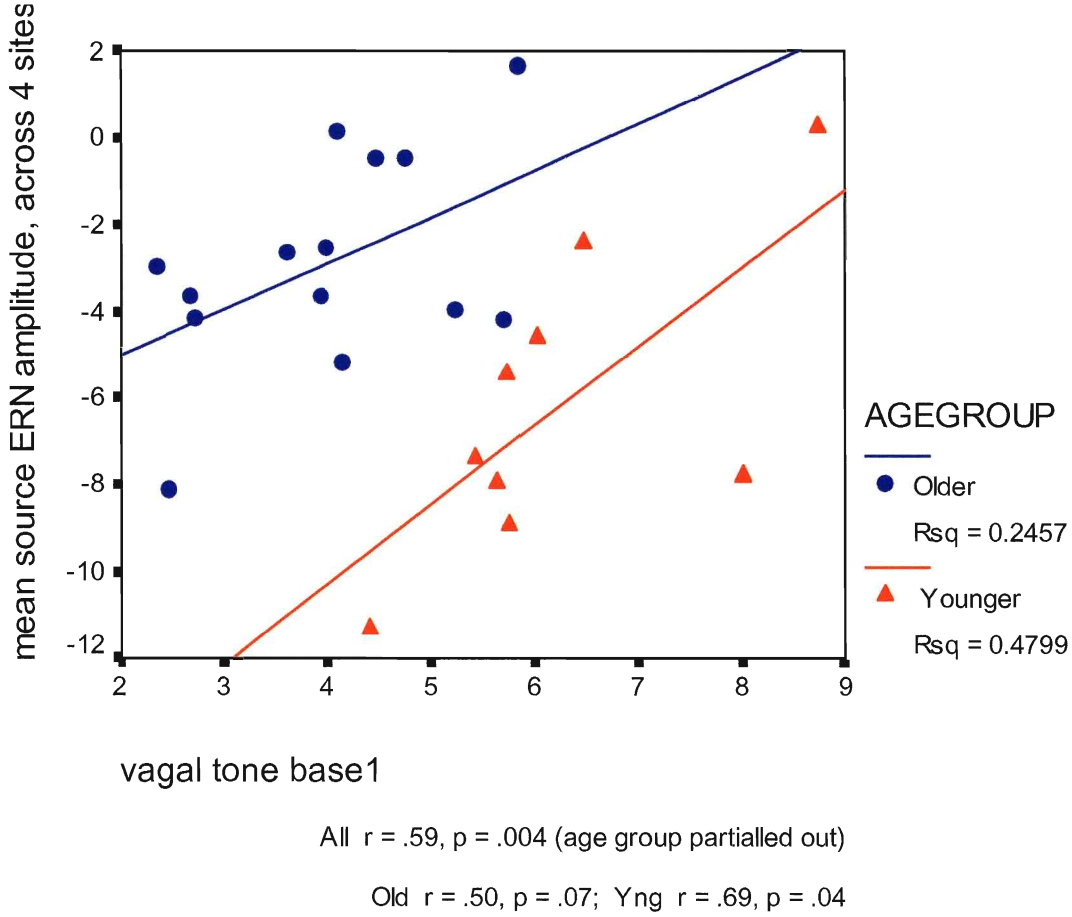


Figure 31. Prediction of mean ERN amplitude from the source memory task by residual vagal tone (VTresid) from Block 1. (N = 22).

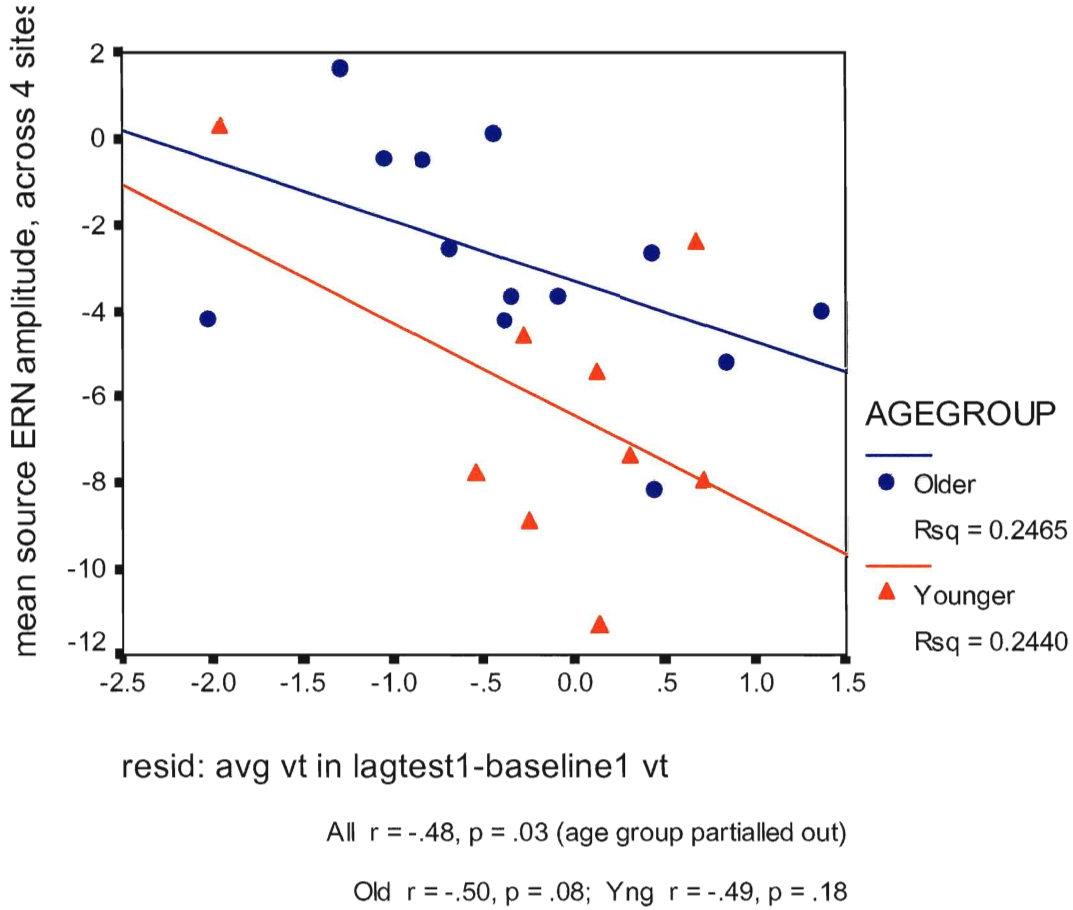


Figure 32. Prediction of mean Pe amplitude from the source memory task by baseline vagal tone. (N = 23).

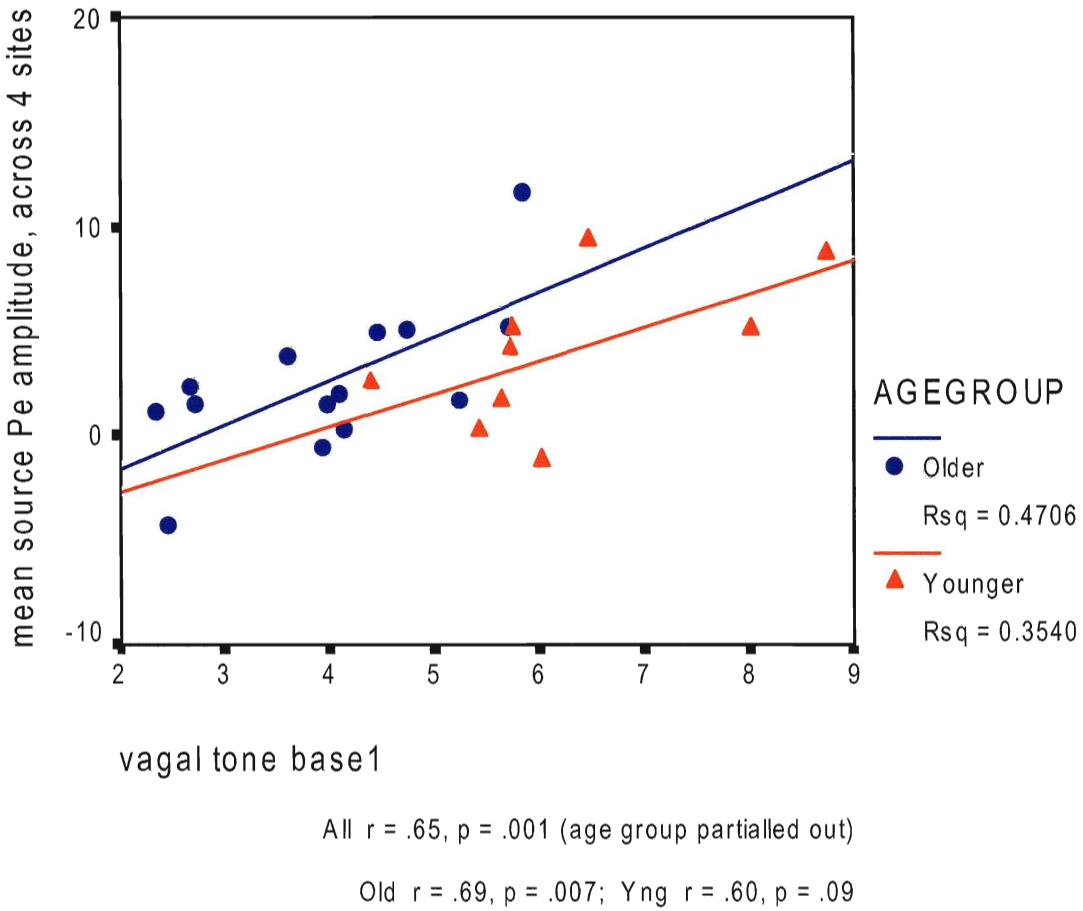
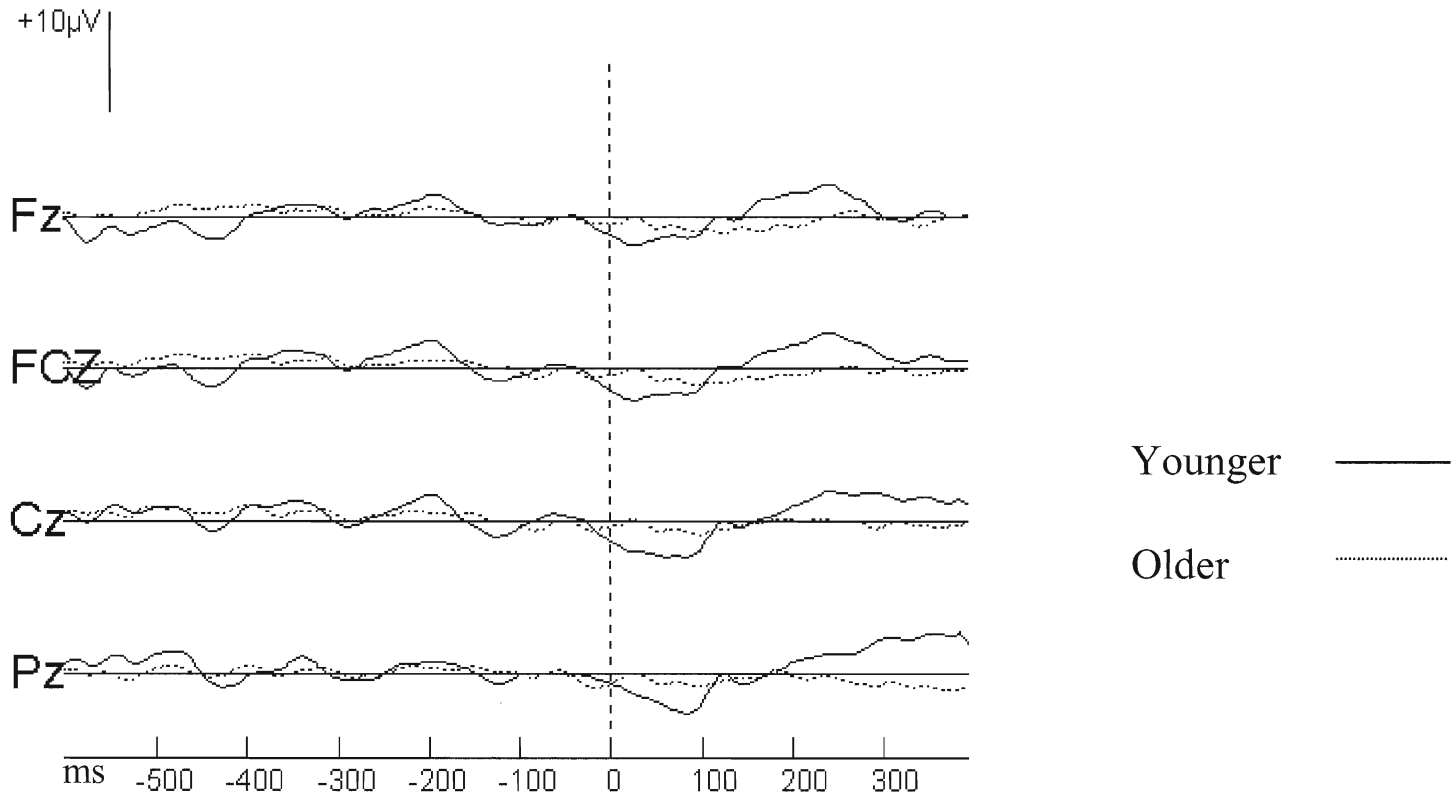


Figure 33. Grand average ERP waveforms (unfiltered) for error trials in the source memory task after subtracting waveforms for correct trials (Difference wave for younger and older adults).



Appendix A: Parasympathetic Control of Cardiac Function.

Appendix A: Parasympathetic Control of Cardiac Function.

The heart is designed to beat spontaneously. The locus of the heart's automaticity is the sino-atrial node (S-A node), at the junction of the superior vena cava and the right atrium. Because the resting potential of cells there (-60 mV) is less negative than that of other myocardium (-70 mV), this area fires more readily than any other heart tissue. The spontaneous rate of discharge of the S-A node is influenced by competing innervation from both the parasympathetic nervous system (via efferent fibres of the vagus nerve) and the sympathetic nervous system. In mammals, the vagus has branches originating in two pairs of nuclei within the medulla, the dorsal medial nucleus and the nucleus ambiguus. These nuclei directly affect the heart to adapt to a variety of situations as needed. For example, when no environmental challenge is present, increased firing of DMN vagal efferents slows heart rate in order to support visceral homeostatic functions as necessary. When a novel change in the environment presents itself, the NA branch increases its efferent action to temporarily slow heart rate to support attentional processes. This branch innervates the soft palate, larynx, pharynx, and bronchi, so has a role in mediating vocalizations in response to changing situations. If dangerous or stressful circumstances arise, however, efferent activity from both vagal branches is withdrawn and the sympathetic system increases heart rate and breathing to enable locomotion for a fight or flight response. Large-scale behavioural responses require an integration of all of the mechanisms of cardiorespiratory control, both sympathetic and parasympathetic (Spyer, 1994). Such integration is the province of the central nervous system (CNS), which must organize patterns of cardiovascular response that are appropriate for the behavioural context of the individual. This includes determining when homeostatic reflexes might have to be modified or abrogated (Hilton, 1966).

The S-A node's intrinsic rate of firing is about 105 beats per minute (bpm), but the average resting heart rate of a seated adult is only 60-80 bpm (Brownley, Hurwitz & Schneiderman, 2000). Like a brake that prevents an automatic car from moving when the engine is running, parasympathetic control of the S-A node keeps heart rate in check. The withdrawal of vagal efferent activity is like easing up on the brake, i.e. it amounts to a cessation of interference with the spontaneous firing of the S-A node, which allows the heart to beat faster (see Katona & Jih, 1975). Berntson et al. (1994) have noted that the dynamic range of the parasympathetic system is much greater than that of the sympathetic system. Moreover, under resting conditions, autonomic tone has been shown to contain about three times as much parasympathetic influence as sympathetic. Thus, primary control of heart rate is accomplished by the parasympathetic system.

Respiratory Sinus Arrhythmia (RSA) Indexes Parasympathetic Function

Measurement of parasympathetic influence is possible because the activity of the vagus nerve is proportional to the peak-to-peak variations in heart period caused by spontaneous respiration, which it mediates (Brownley et al., 2000; Fracasso, Porges, Lamb & Rosenberg, 1994; McDonald, 1980). During inspiration, cardiac vagal motor neurons are inhibited; as a result, heart period is smaller, the heart rate quickens. Conversely, during expiration, vagal action is reinstated as cardiac vagal motor neurons discharge (Spyer, 1994) causing heart period to lengthen, and heart rate to slow. This

effect is known as respiratory sinus arrhythmia (RSA; Brownley et al. 2000; Fracasso, Porges, Lamb & Rosenberg, 1994; McDonald, 1980). Since these oscillations are vagally mediated, RSA provides an estimate of vagal function. Because vagal efferent activity is yoked to respiration, there must be a direct connection between respiratory and cardiovascular function, likely a common source nucleus in the medulla (Katona & Jih, 1975; Spyer, 1994; Brownley et al., 2000). The right and left branches of the vagus are not identical (Hermanowicz & Truong, 1999). According to Porges, the right branch of the NA vagus is the principal determinant of RSA (Porges, Doussard-Roosevelt & Maiti, 1994).

Blockade of vagal function can be accomplished by administration of the drug atropine (e.g. Berntson et al., 1994). Vagal blockade virtually eliminates the heart rate variability that is due to RSA from the cardiac signal, making the signal simpler. In the case of cardiac function, simpler is not better. Greater complexity in the cardiac signal wave indicates better physical health (Fallen, 2000). Complex signals indicate that afferent influences on heart rate from a variety of physiological functions are successfully reaching the brain, being processed there, and influencing it to adjust heart rate in proportion to the body's needs. Toward the end stages of many diseases, however, cardiac signals become much simpler, almost like sine-waves, indicating a drastic loss of physiologic regulation (Goldberger, 1996). In these circumstances, the heart is not able to adapt appropriately to constantly changing conditions.

Through specialized techniques, RSA is detectable as a small fluctuation in the heart rate signal. The amplitude of the hidden respiratory fluctuations in resting RSA is sometimes referred to as vagal "tone". However, RSA is the result of both tonic and phasic components of vagal activity. These two highly interdependent aspects are nevertheless represented by distinct functions and behavioural changes (Berntson, Bigger, Eckberg, et al., 1997). Tonic vagal activity reflects an individual's idiosyncratic reactivity or ability to maintain an organized behavioural state and visceral homeostasis (Porges, Doussard-Roosevelt, Portales & Greenspan,1996) and may be thought of as characteristic of an individual's resting state. Conversely, *phasic* vagal changes index the flexibility with which vagal efferent activity may be up-regulated or down-regulated in response to situational or task demands (Porges, et al. 1996; Huffman, Bryan, del Carmen, et al., 1998). In order to investigate these influences separately, Huffman et al. recommend that vagal efferent activity be calculated from heart rate data taken both at rest and during social or cognitive challenges.

Despite the utility of RSA as an index of vagal function, spectral analyses of the power of the cardiac signal reveal that the rhythmic variations due to RSA represent only a small portion of the entire spectral energy. Most of the spectral energy (about 80%) is related to the control of mean arterial pressure (MAP) and mediated sympathetically (Mulder & Mulder, 1981). The variance in heart rate that is attributable to vagal activity affiliated with respiration is less than 20%, and can be as small as 1% in a fetus (Donchin, Caton & Porges, 1984, as quoted in Porges & Bohrer, 1990). To "see" such small oscillations, one must remove from the cardiac signal the slow, complex trends that are due to large-scale influences such as intermittent sympathetic efferent activity. This cannot be done by linear detrending, nor can it be done properly by spectral analysis unless the data fulfill certain assumptions (e.g., stationarity) which is often impossible

with normally complex physiological data (Porges & Bohrer, 1990). For example, heart period is the final outcome of multiple periodic physiological processes. In 1985, Porges patented a unique time-series method whereby the amplitude of RSA may be quantified and used as an estimate of parasympathetic (vagal) activity. By moving a polynomial filter step-wise through the complex cardiac signal, low-frequency trends can be removed, allowing remaining higher frequency physiological activity, such as RSA, to be quantified accurately. Our lab derives calculations of vagal activity based on this method.

RSA measures in particular are increasingly being used to examine neurological and psychological correlates of behaviour in psychophysiological research (Brownley et al., 2000; Weber et al., 1994; Porges, 1995a, 1985; Mulder & Mulder, 1981). RSA is reduced with age (McDonald, 1980; Taylor, Hayano, & Seals, 1995). Compromised heart rate variability may help explain the attentional problems that older adults have (van der Molen, Somsen & Jennings, 1996; see also Fallen, 2000), problems that may be manifested in greater numbers of errors, slower responses and even radical failure to complete a task (Band & Kok, 2000).

Appendix B: Interpretation of the ERN.

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A variety of opinions exist regarding the meaning of the ERN: it may represent response conflict (Carter et al., 1998); error-detection (Falkenstein et al., 1991); response monitoring (Falkenstein et al., 2000), some kind of emotional response to an error (Gehring & Knight, 2000; Vidal, Hasbroucq, Grapperon & Bonnet, 2000; Falkenstein et al., 2000) or even the ACC's designation of a neural command centre to mobilize the motor system to deal with unexpected negative consequences (Holroyd & Coles, 2002).

Reports have come in of finding small negativities in ERP responses to *correct* trials, (dubbed "CRNs") although they are smaller than the negativity generated on error trials. If correct trials are capable of inducing negative deflections, however, then the ERN cannot be simply a manifestation of error-detection, although it might reflect a process that happens concurrently with errors, such as response monitoring. Because some negativity occurs to correct as well as incorrect responses, a process 'upstream' from actual detection of the error might be responsible for the ERN (Vidal et al., 2000; Falkenstein et al., 2000, 2001).

It is also possible to encounter conscious awareness of errors unaccompanied by error negativity in the electrophysiological data (Stemmer et al., 2001). Since the ERN may be dissociated from awareness of errors, response checking may be a plausible explanation for the ERN, although the evidence for this is still preliminary (Falkenstein et al., 2000).

The ERN is attenuated when responses are made impulsively, before processing is complete, or under conditions of time pressure (Falkenstein et al., 1991). It is shallower when the memory load for a task is higher (Scheffers, Humphrey, Stanny, et al., 1999); when there is uncertainty about having committed an error (Scheffers, & Coles, 2000), or errors are harder to detect (Falkenstein et al., 1996); when the response is correct, or when participants produce only partial, EMG errors, (Vidal et al., 2000; Falkenstein, et al., 2000; see also Carter et al., 1998 and Gehring and Knight, 2000); when participants are sleep-deprived after a long time on task (Scheffers et al., 1999); among participants who have been poorly socialized (Dikman & Allen, 2000); when an error has been committed deliberately, i.e., it has been faked and thus there is no "error" from the point of view of the person (Segalowitz, TENNET XII conference, 2001) or when it has been committed "honestly", i.e. the person has not realized that his or her choice was incorrect and thus detected no error (Scheffers, Coles, Bernstein, et al., 1996). It has also been seen to be smaller among older adults and among patients with Parkinson's disease (Falkenstein et al., 2001).

Conversely, the ERN has been shown to have a larger amplitude under a variety of opposite conditions. ERN amplitude is large when impulsive responding is low, i.e. when participants strive for accuracy (Miltner et al., 1997; Gehring et al., 1993); when errors are easy to detect and participants are certain when they have made an error (Scheffers & Coles, 2000; Falkenstein et al., 2000); when the error was important to the participant, i.e. he or she made "a big mistake"(Falkenstein et al., 2000); when two kinds of errors (choice and action slip) were committed at the same time, (Bernstein et al., 1995); and when the probability of distracting information surrounding the target is high (Falkenstein, TENNET XII conference, 2001). Because OCD patients have been shown

to generate large ERNs (Gehring, Himle & Nisenson, 2000), some researchers have hypothesized that OCD may be partly a result of a hyper-sensitive error-detection system.

Despite numerous studies, no consensus on the interpretation of the ERN has yet been reached. It seems however, that its amplitude varies with the importance of the error to the participant.

Appendix C: Interbeat Interval Analyses

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As mentioned, considerable differences in VT were expected between the younger and older groups, which were indeed found in the present data set. These group differences in variability might be real and substantive differences or they might be explained simply by higher mean scores in the young group. To address this issue, the coefficients of variation (CV = SD/IBI) were examined for each group. Generally, the CV provides an indication of the variability for a given *level* of a measure such as the interbeat interval (IBI). If the CVs for each group are the same, group differences in variability, i.e. in the groups' standard deviations (SD), can simply be explained by higher scores in one of the groups. If this is the case, differences between the groups are only quantitative (Segalowitz & Segalowitz, 1993; Howell, 1997). If the CVs for the groups are different, however, then the difference in variability between the two groups (i.e., the difference in the SD of the IBI, or in VT), may be relied on as a real and substantive difference that distinguishes the groups.

Several cardiac measures were derived from the ECG for each of the time periods for which vagal tone was calculated. These included VT, mean interbeat interval (IBI), the IBI standard deviation (SD), and the coefficient of variation (CV). In order to determine whether IBI or vagal tone was the more sensitive measure of group differences, first, each of the other cardiac measures (IBI, SD and CV) was entered into a correlational analysis with VT, controlling for age group. The correlational analyses were carried out using Baseline 1 and average measures for each task (flanker, Source Block 1, Source Block 2). Second, a similar set of analyses correlated IBI measures with SD, CV and VT.

Baseline 1 (resting) VT correlated positively with baseline SD ($r = .71$, $p < .001$) and baseline CV ($r = .70$, $p < .001$), but not with baseline IBI ($r = .32$, $p = .09$; see Table 10). During tasks, the relationships between VT and the other cardiac variables changed slightly. In the flanker task, VT correlated positively with all other cardiac variables, i.e., with CV ($r = .66$, $p < .001$), with SD ($r = .67$, $p < .001$) and with IBI ($r = .39$, $p = .04$) measures from this period. The situation for the source memory task was similar: Block 2 VT correlated with CV ($r = .67$, $p < .001$), SD ($r = .70$, $p < .001$) and also IBI ($r = .45$, $p = .02$) from Block 2. Block 1 VT correlated with Block 1 CV ($r = .78$ $p < .001$) and SD ($r = .76$ $p < .001$), but not IBI ($r = .33$, $p = .10$).

It should be noted that during Baseline 1, IBI itself correlated positively with SD ($r = .45$, $p = .02$; see Table 11), indicating that before any tasks had begun, heart rate and *overall* variability in heart rate were related. In addition, during tasks, IBI usually correlated with SD. (The exception was during Block 1 of the source memory task). However, IBI never correlated with CV in any condition. Thus, vagal tone measures from ECG data collected during task appeared to capture variance that was different from that captured by IBI.

Although a relative dearth of overall relationships between IBI and other cardiac measures appeared in both groups, this was especially obvious in the case of the older adults. In this group, none of the IBI variables correlated with any of the other cardiac variables – including VT measures – either at baseline or during task performance. In contrast, for the younger group, IBI always correlated with VT measures, and also with SD during Block 2 of the source memory task. Additionally, baseline IBI correlated with

all of the other baseline cardiac variables, for the younger adults. The correlations for the groups separately are presented in Table 12 (VT with other variables) and Table 13 (IBI with other variables).

Vagal tone measures also appeared to be more sensitive than IBI in distinguishing between groups. T-tests revealed no group differences in baseline IBI measures, $t(27) = .74$, n.s., nor in IBI measures during any of the task conditions. (Refer to Table 14 for a list). On the other hand, VT, SD, and CV showed strong group differences in every condition. Therefore, it may be said that vagal tone was confirmed as a more sensitive measure of cardiac control than IBI. Finally, since significant group differences were found in the CV measures, the differences in variability (i.e., in VT and SD) between the younger and older group were taken to be real and substantive.

It should not be surprising that IBI does not always correlate with vagal tone, since, despite being cardiac measures derived from the same ECG files, IBI and vagal tone quantify two different things. IBI, the inverse of heart rate, as the outcome of many influences on the heart, is a fairly gross measure. Vagal tone, on the other hand, specifically reflects RSA, the small, regular fluctuations in heart rate that are yoked to respiration rate. At rest, these two measures might be expected to be correlated. But since attentional demands in paradigms such as the flanker and lag tasks produce a withdrawal of vagal tone, and an increase in heart rate, it might be suspected that other influences–such as the sympathetic system–have a role in sustaining a faster heart rate, in order to support the effort required by the task

Vagal Tone and IBI Analyses. Although the principal cardiac measure of interest in this study is vagal tone, because VT is a calculated construct whose effects are sometimes difficult to envision, analyses of interbeat intervals (IBIs) have been carried out that parallel those involving vagal tone. It is hoped that these IBI analyses will allow the reader to understand what general changes take place in the heart rate itself, across baselines, across tasks, and from baseline to task. Increases in IBI represent decreases in heart rate and vice versa. Because IBI and vagal tone measure different aspects of cardiac behaviour (rate versus respiratory-related variability, respectively), the results of the analyses are not identical.

Heart rate data analogous to the vagal tone data from the baseline periods were submitted to a repeated measures ANOVA, with baseline period as the within-group factor and group as the between-group factor. This revealed a corresponding increase in IBI, $F(2, 54) = 6.68$, $p = .006$, $\eta^2 = .198$, indicating that heart rate slowed overall across the baseline conditions, periods in which participants relaxed before or after tasks. This result implies complete recovery of resting heart rate after tasks were completed, in both groups. There was no interaction with group. In contrast to the vagal tone analysis, the between-subjects difference in IBI was not significant.

IBI data from the flanker task were submitted to a repeated measures ANOVA with flanker period as the within-group factor and group as the between-group factor. In contrast to the analysis of vagal tone for the flanker task, the between-subjects difference was not significant. This revealed an overall increase in heart rate across the flanker task (IBI: $M = 847$ ms to 823 ms; $F(2, 54) = 6.02$, $p = .017$, $\eta^2 = .18$). There was no interaction with group.

IBI data submitted to a repeated measures ANOVA with source memory period as the within-group factor (Block1, Block 2) and age (older vs. younger) as the between-group factor. Again, in contrast to the significant age effect in the analysis of VT during the source memory task, there was no age effect in this IBI analysis, $F(1, 26) = .65$, n.s. However, the analysis did reveal changes in mean IBI across the source memory task, $F(1, 26) = 10.61$, $p = .003$, $\eta^2 = .29$. Heart rate in Block 1 (*M* IBI = 847 ms) was faster than in Block 2 (*M* IBI = 868 ms). Overall heart rate at the start of the second block was as slow as it had been at baseline (*M* IBI = 867 ms) although this effect was mainly due to the younger group (younger *M* IBI = 889 ms vs. older *M* IBI = 847 ms). Block 2 of the source memory paradigm is the only task period during which heart rate was actually as low as in baseline levels.

Appendix D: Analysis Summary Tables

TableD-1.
2 (Congruency Condition) x 2 (Group) Mixed ANOVA for Per cent Errors in the Flanker Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Congruency (C)	.028	1	37.01	.000	.58
C x G	.0001	27	.17	.68	.01
Error	.021	27			
Between-Subjects Effects					
Group (G)	.018	1	5.11	.03	.16
Error	.094	27			

Table D-2.
2(Congruency Condition) x 2 (Group) Mixed ANOVA for Response Times to Correct Trials in the Flanker Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Congruency (C)	10610.23	1	104.93	.000	.80
C x G	652.70	1	6.46	.02	.19
Error	2730.06	27			
Between-Subjects Effects					
Group (G)	48585.49	1	10.92	.003	.29
Error	120150.19	27			

Table D-3.
4 (Site) x 2 (Group) Mixed ANOVA for Amplitude of P3 from Error Trials in the Flanker Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Site (S)	104.94	3	5.81	.01	.20
S x G	131.14	3	7.26	.005	.24
Error	415.33	69			
Between-Subjects Effects					
Group (G)	121.15	1	1.18	.29	.05
Error	2362.35	23			

Table D-4.
2 (Congruency Condition) x 4 (Site) x 2 (Group) Mixed ANOVA for ERN Amplitude to Error Trials in the Flanker Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Congruency (C)	51.12	1	.71	.41	.02
C x G	149.15	1	2.06	.16	.06
Error	2175.46	30			
Site (S)	507.25	3	16.67	.000	.36
S x G	397.77	3	13.08	.000	.30
Error	912.67	90			
C x S	6.66	3	.67	.53	.02
C x S x G	18.01	3	1.81	.17	.06
Error	298.67	90			
Between-Subjects Effects					
Group (G)	1070.93	1	6.38	.02	.18
Error	5033.59	30			

Note. ERN amplitudes were determined in relation to a baseline from 600 to 400 ms prior to the response.

Table D-5.
4 (Site) x 2 (Group) Mixed ANOVA for Amplitude of ERN to Error Trials in the Flanker Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Site (S)	387.98	3	23.09	.000	.50
S x G	198.25	3	11.80	.000	.34
Error	386.43	69			
Between-Subjects Effects					
Group (G)	815.29	1	12.05	.002	.34
Error	1555.87	23			

Note. ERN amplitude was collapsed across congruent and incongruent trials and determined in relation to a baseline from 600 to 400 ms prior to the response.

Table D-6.
4 (Site) x 2 (Group) Mixed ANOVA for Amplitude of Difference-Wave ERN for Flanker Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Site (S)	383.50	3	23.25	.000	.44
S x G	66.84	3	4.05	.03	.12
Error	494.86	90			
Between-Subjects Effects					
Group (G)	996.25	1	9.99	.004	.25
Error	2991.74	30			

Note. ERN amplitude was collapsed across congruent and incongruent trials and determined in relation to a baseline from 600 to 400 ms prior to the response.

Table D-7.
2 (Congruency Condition) x 4 (Site) x 2 (Group) Mixed ANOVA for Pe Amplitude to Error Trials in the Flanker Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Congruency (C)	417.69	1	9.31	.005	.24
C x G	85.79	1	1.91	.18	.06
Error	1346.23	30			
Site (S)	316.00	3	12.84	.000	.30
S x G	283.80	3	11.53	.000	.28
Error	738.32	90			
C x S	3.40	3	.36	.72	.01
C x S x G	3.88	3	.41	.68	.01
Error	284.22	90			
Between-Subjects Effects					
Group (G)	1070.93	1	6.38	.02	.18
Error	5033.59	30			

Note. Pe amplitudes were determined in relation to a baseline from 600 to 400 ms prior to the response.

Table D-8.
4 (Site) x 2 (Group) Mixed ANOVA for Amplitude of Pe in the Flanker Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Site (S)	111.19	3	15.27	.000	.40
S x G	129.50	3	17.78	.000	.44
Error	167.50	69			
Between-Subjects Effects					
Group (G)	643.09	1	8.52	.008	.27
Error	1736.24	23			

Note. Pe amplitude was collapsed across congruent and incongruent trials and determined in relation to a baseline from 600 to 400 ms prior to the response.

Table D-9.
4 (Site) x 2 (Group) Mixed ANOVA for Amplitude of Difference-Wave Pe in the Flanker Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Site (S)	286.12	3	31.83	.000	.52
S x G	156.12	3	17.37	.000	.37
Error	269.65	90			
Between-Subjects Effects					
Group (G)	909.51	1	7.77	.009	.21
Error	3509.95	30			

Note. Pe amplitude was collapsed across congruent and incongruent trials and determined in relation to a baseline from 600 to 400 ms prior to the response.

Table D-10.
3 (Word-type) x 2 (Group) Mixed ANOVA for proportion of "Yes" Responses for Source Memory Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Word-type (W)	3.64	2	165.85	.000	.85
W x G	.67	2	30.66	.000	.51
Error	.66	60			
Between-Subjects Effects					
Group (G)	.30	1	7.10	.01	.19
Error	1.26	30			

Table D-11.
3 (Word-type) x 2 (Group) Mixed ANOVA for Response Times to Correct Trials in the Source Memory Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Word-type (W)	86095.55	2	2.07	.15	.07
W x G	85327.39	2	2.05	.15	.06
Error	1247962.99	60			
Between-Subjects Effects					
Group (G)	1004882.68	1	4.82	.04	.14
Error	6258815.86	30			

Table D-12.
3 (Word-type) x 4 (Site) x 2 (Group) Mixed ANOVA Mean LP Areas for Source Memory Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Word-type (W)	182.05	2	8.74	.000	.23
W x G	86.05	2	4.13	.021	.12
Error	624.78	60			
Site (S)	74.70	3	7.21	.003	.19
S x G	170.20	3	16.43	.000	.35
Error	310.87	90			
W x S	3.19	6	1.16	.33	.04
W x S x G	1.15	6	.42	.70	.01
Error	82.61	180			
Between-Subjects Effects					
Group (G)	57.85	1	.73	.40	.02
Error	2384.42	30			

Note. LP amplitudes were determined from stimulus-locked trials.

Table D-13.
4 (Site) x 2 (Group) Mixed ANOVA for Amplitude of ERN in Source Memory Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Site (S)	31.36	3	6.68	.003	.23
S x G	7.18	3	1.53	.23	.06
Error	107.93	69			
Between-Subjects Effects					
Group (G)	270.22	1	8.43	.008	.27
Error	737.52	23			

Note. ERN amplitude was determined in relation to a baseline from 200 to 0 ms prior to the response.

Table D-14.
4 (Site) x 2 (Group) Mixed ANOVA for Amplitude of Pe in the Source Memory Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Site (S)	151.34	3	15.41	.000	.40
S x G	74.77	3	7.61	.002	.25
Error	225.85	69			
Between-Subjects Effects					
Group (G)	37.41	1	.78	.39	.03
Error	1105.03	23			

Note. Pe amplitude was determined in relation to a baseline from 200 to 0 ms prior to the response.

Table D-15.
2 (Paradigm) x 4 (Site) x 2 (Group) Mixed ANOVA for Comparison of P3 (Flanker Task) with LP (Source Memory Task).

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Paradigm (P)	552.51	1	13.74	.001	.31
P x G	88.31	1	2.20	.15	.07
Error	1206.05	30			
Site (S)	86.11	3	7.54	.003	.20
S x G	163.02	3	14.27	.000	.32
Error	342.65	90			
P x S	29.64	3	2.71	.10	.08
P x S x G	6.47	3	.59	.50	.02
Error	328.48	90			
Between-Subjects Effects					
Group (G)	1.57	1	.02	.90	.00
Error	2834.20	30			

Note. P3 was determined from response-locked error trials. LP was determined from stimulus-locked correct trials.

Table D-16.
2 (Paradigm) x 4 (Site) x 2 (Group) Mixed ANOVA for Comparison of ERN (Flanker Task) with ERN (Source Memory Task).

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Paradigm (P)	17.79	1	.28	.60	.01
P x G	73.39	1	1.17	.29	.05
Error	1448.07	23			
Site (S)	308.35	3	27.60	.000	.55
S x G	96.04	3	8.60	.001	.27
Error	257.00	69			
P x S	110.98	3	10.75	.000	.32
P x S x G	109.39	3	10.60	.000	.32
Error	237.36	69			
Between-Subjects Effects					
Group (G)	1012.13	1	27.54	.000	.55
Error	845.34	23			

Table D-17.
2 (Paradigm) x 4 (Site) x 2 (Group) Mixed ANOVA for Comparison of Pe (Flanker Task) with Pe (Source Memory Task).

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Paradigm (P)	917.53	1	12.51	.002	.35
P x G	185.14	1	2.53	.13	.10
Error	1687.22	23			
Site (S)	157.70	3	20.73	.000	.47
S x G	18.30	3	2.41	.09	.10
Error	174.97	69			
P x S	104.83	3	11.04	.000	.32
P x S x G	185.97	3	19.59	.000	.46
Error	218.37	69			
Between-Subjects Effects					
Group (G)	495.36	1	9.87	.005	.30
Error	1154.05	23			

Table D-18.
3 (Time-Period) x 2 (Group) Mixed ANOVA for Baseline Vagal Tone.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Time-Period (T)	3.40	2	6.36	.003	.19
T x G	.08	2	.16	.85	.01
Error	14.44	54			
Between-Subjects Effects					
Group (G)	109.18	1	31.58	.000	.54
Error	93.33	27			

Table D-19.
3 (Time-Period) x 2 (Group) Mixed ANOVA for Vagal Tone During the Flanker Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Time-Period (T)	1.74	2	3.14	.06	.11
T x G	.03	2	.05	.93	.00
Error	14.36	52			
Between-Subjects Effects					
Group (G)	92.07	1	29.93	.000	.54
Error	79.98	26			

Table D-20.
2 (Block) x 2 (Time-Period) x 2 (Group) Mixed ANOVA for Vagal Tone During the Source Memory Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Block (B)	7.93	1	21.71	.000	.47
B x G	.41	1	1.14	.30	.04
Error	9.13	25			
Time-Period (T)	.40	1	2.67	.12	.10
T x G	.12	1	.81	.38	.03
Error	3.74	25			
B x T	.01	1	.05	.83	.002
B x T x G	.07	1	.40	.54	.02
Error	4.75	25			
Between-Subjects Effects					
Group (G)	170.81	1	38.40	.000	.61
Error	111.22	25			

Table D-21.
2 (Paradigm) x 2 (Group) Mixed ANOVA for Comparison of Vagal Tone Levels During the Flanker Task with Vagal Tone Levels During Block 1 of the Source Memory Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Paradigm (P)	.04	1	.24	.63	.01
P x G	.48	1	3.27	.08	.11
Error	3.83	26			
Between-Subjects Effects					
Group (G)	72.74	1	33.57	.000	.56
Error	56.34	26			

Table D-22.
2 (Paradigm) x 2 (Group) Mixed ANOVA for Residual Vagal Tone from the Flanker Task and Block 1 of the Source Memory Task.

	ANOVA Summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subjects Effects					
Paradigm (P)	.0001	1	.001	.98	.000
P x G	.01	1	.11	.75	.004
Error	3.61	26			
Between-Subjects Effects					
Group (G)	4.23	1	2.96	.10	.10
Error	37.12	26			

Note. To produce the residual vagal tone variable, Baseline 1 vagal tone has been covaried from task-level vagal tone from both tasks.

Table D-23.
Hierarchical Regression Analysis Predicting Source Memory Task Errors for Group (older vs. younger adults) from Baseline 3 Vagal Tone (B3).

Predictor	*β*	*pr*	*sr*	*Cum* R^2	*Inc* R^2	*F*	*df*	*p*
Step 1								
Group (G)	.68***	.68	.68	.47	.47	23.52	1, 27	.000
Step 2								
B3	-.55**	-.49	-.36	.59	.13	8.12	1, 26	.008
Step 3								
G x B3	-.58	-.22	-.14	.61	.02	1.22	1, 25	.28

** $p < .01$, *** $p < .001$. F statistics refer to significance of the increment in R^2 (Inc. R^2) at each step. Cum. R^2 = cumulative R^2 to that point in the equation.

Table D-24.
Hierarchical Regression Analysis Predicting Error Positivity (Pe) for Group (older vs. younger adults) from Residualized Vagal Tone (VTr) in the Flanker Task.

Predictor	*β*	*pr*	*sr*	*Cum* R^2	*Inc* R^2	*F*	*df*	*p*
Step 1								
Group (G)	-.52**	-.52	-.52	.27	.27	9.74	1, 26	.004
Step 2								
VTr	.37*	.42	.36	.40	.13	5.28	1, 25	.03
Step 3								
G x VTr	.16	.06	.05	.40	.00	.10	1, 24	.76

* $p < .05$, ** $p < .01$. F statistics refer to significance of the increment in R^2 (Inc. R^2) at each step. Cum. R^2 = cumulative R^2 to that point in the equation. Baseline 1 vagal tone has been covaried from on-task vagal tone during flanker task.

Table D-25.
Hierarchical Regression Analysis Predicting Error Negativity (ERN) for Group (older vs. younger adults) from Baseline 1 Vagal Tone (B1) in the Source Memory Task.

Predictor	*β*	*pr*	*sr*	*Cum* R^2	*Inc* R^2	*F*	*df*	*p*
Step 1								
Group (G)	.50*	.50	.50	.25	.25	6.94	1, 21	.02
Step 2								
B1	.70**	.59	.51	.51	.26	10.80	1, 20	.004
Step 3								
G x B1	-.49	-.19	-.14	.53	.02	.75	1, 19	.40

* $p < .05$; ** $p < .01$. F statistics refer to significance of the increment in R^2 (Inc. R^2) at each step. Cum. R^2 = cumulative R^2 to that point in the equation.

Table D-26.
Hierarchical Regression Analysis Predicting Error Negativity (ERN) for Group (older vs. younger adults) from Residualized Vagal Tone (VT1r) in Block 1 of the Source Memory Task.

Predictor	*β*	*pr*	*sr*	*Cum* R^2	*Inc* R^2	*F*	*df*	*p*
Step 1								
Group (G)	.49*	.49	.49	.24	.24	6.45	1,20	.02
Step 2								
VT1r	-.42*	-.48	-.42	.42	.18	5.80	1,19	.03
Step 3								
G x VT1r	.34	.12	.09	.43	.01	.27	1,18	.61

* $p < .05$. F statistics refer to significance of the increment in R^2 (Inc. R^2) at each step. Cum. R^2 = cumulative R^2 to that point in the equation. Baseline 1 vagal tone has been covaried from on-task vagal tone during block 1 of the source memory task.

Table D-27.
Hierarchical Regression Analysis Predicting Error Positivity (Pe) for Group (older vs. younger adults) from Baseline 1 Vagal Tone (B1) in the Source Memory Task.

Predictor	β	*pr*	*sr*	*Cum* R^2	*Inc* R^2	*F*	*df*	*p*
Step 1								
Group (G)	-.20	-.20	-.20	.04	.04	.89	1,21	.36
Step 2								
B1	.87***	.65	.64	.44	.40	14.34	1,20	.001
Step 3								
G x B1	.33	.12	.09	.45	.01	.29	1,19	.60

*** $p \leq .001$. F statistics refer to significance of the increment in R^2 (Inc. R^2) at each step. Cum. R^2 = cumulative R^2 to that point in the equation.

Table D-28.
Hierarchical Regression Analysis Predicting Difference Wave Error Negativity (ERN) for Group (older vs. younger adults) from Baseline 1 Vagal Tone (B1) in the Source Memory Task.

Predictor	*β*	*pr*	*sr*	*Cum* R^2	*Inc* R^2	*F*	*df*	*p*
Step 1								
Group (G)	.35	.35	.35	.12	.12	2.80	1,20	.11
Step 2								
B1	.57*	.45	.42	.30	.18	4.85	1,19	.04
Step 3								
G x B1	.10	.03	.03	.30	.00	.02	1,18	.89

* $p \leq .05$. F statistics refer to significance of the increment in R^2 (Inc. R^2) at each step. Cum. R^2 = cumulative R^2 to that point in the equation.

Appendix E: Source Memory Task Stimuli

Table E-1.
Word Stimuli for the Source Memory Task

Set 1	*Set2*	*Set3*	*Set4*	*Set5*	*Set 6*	*Set 7*	*Set 8*
later	peace	table	sound	alone	black	third	value
moved	power	light	large	thing	along	began	least
green	wheel	truck	theme	truly	alive	uncle	apart
swung	fixed	touch	aware	spoke	shape	enemy	sight
front	sleep	fifty	dress	sweet	aside	drawn	quick
ideal	added	basic	range	stage	river	union	shown
built	solid	model	frame	judge	knife	enter	title
tired	given	young	often	group	among	point	order
cover	route	pilot	pride	occur	agent	grown	brush
fully	award	limit	curve	royal	grand	blind	guard
sense	taken	today	money	local	whose	above	field
space	enjoy	prior	steel	grant	trend	catch	threw
whole	dance	equal	worth	break	order	carry	civil
stock	event	index	drink	round	fresh	watch	train
cloth	phase	sharp	brief	upper	allow	forth	legal
clean	story	issue	paper	earth	final	stand	start
offer	shook	exist	stone	scale	avoid	shore	taste
dozen	hoped	trust	anger	raise	chain	sorry	angle
check	staff	seven	speak	plane	heavy	doubt	faith
mouth	novel	dream	phone	spite	inner	motor	cross
metal	level	child	total	party	stood	music	short
apply	claim	glass	share	meant	happy	noted	fight
guess	depth	vital	block	empty	chair	coast	score
woman	close	court	known	force	south	voice	board
unity	scene	drive	price	eight	serve	spent	reach